# 6. Baseline Risk Assessment

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# 6. BASELINE RISK ASSESSMENT

The human health risk assessment approach used in the WAG 5 baseline risk assessment (BRA) was based on the EPA *Risk Assessment Guidance for Superfund* (RAGS) (EPA 1989, May 1992), INEEL Track 2 guidance (DOE-ID 1994), and the INEEL cumulative risk assessment guidance protocol (LMITCO 1995). As discussed in the INEEL cumulative risk assessment protocol (LMITCO 1995), the analysis methods used in INEEL comprehensive risk assessments are often different from the analysis methods used in INEEL Track 1 and Track 2 risk assessments (DOE-ID 1992; DOE-ID 1994). In general, the differences between the two types of analyses occur because comprehensive risk assessments are meant to analyze risks produced by multiple sites within a WAG, while Track 1 and Track 2 risk assessments are meant to analyze risks only from one site at a time.

To satisfy the broader objective of INEEL comprehensive risk assessments, analyzing risks produced through the air and groundwater exposure pathways in a "cumulative" manner is recommended in the INEEL cumulative risk assessment guidance protocol (LMITCO 1995). A cumulative analysis of these two exposure pathways involves calculating one risk number for each COPC in each air and groundwater exposure route (e.g., inhalation of fugitive dust and ingestion of groundwater) for each site group. Site groups for WAG 5 are discussed in Section 4. Analyzing the air and groundwater pathways in a cumulative manner is necessary because contamination from all sites within a site group can contribute to air and groundwater contamination for that group. Conversely, individual sites within a WAG are typically isolated from one another relative to the soil pathway exposure routes (e.g., ingestion of soil and of homegrown produce). As a result, soil pathway exposures are analyzed on a site-specific, or "noncumulative," basis in the INEEL comprehensive risk assessments. The details of the "comprehensive" and "cumulative" aspects of the WAG 5 BRA are discussed more completely in the following sections. In general, the BRA is "comprehensive" because it evaluates risks from all known and potential sites within WAG 5, and it is "cumulative" because risks from multiple sites are evaluated in the air and groundwater exposure pathways.

The term "risk" is used throughout this document to refer to the possibility of adverse health effects from either carcinogenic or noncarcinogenic contaminants; however, it also can be used when only carcinogenic health effects are discussed. The term "hazard quotient" is used when only noncarcinogenic health effects are discussed.

## 6.1 Baseline Risk Assessment Tasks

In general, the tasks associated with development of the WAG 5 human health risk assessment include the following:

- Data evaluation
- Exposure assessment
- Toxicity assessment
- Risk characterization.

These tasks are described in the following subsections. As discussed in Section 6.1.4, uncertainty analysis is a component of risk analysis and, therefore, is discussed in Section 6.5.

### 6.1.1 Data Evaluation

All sampling data collected to date at WAG 5 sites (see Section 4 for a discussion of the various WAG 5 sampling investigations) were evaluated to determine whether the data are appropriate and adequate for use in the BRA. This evaluation was conducted generally in accordance with EPA guidance (EPA 1992). As part of this analysis, sampling data sets were assumed to have lognormal distributions in accordance with EPA guidance on calculating concentration terms (EPA 1992). However, true statistical distributions for the data were not determined. A zero concentration for all sampling results below the minimum detection limit (MDL), nondetections, was assumed for the upper confidence limit calculations performed for the BRA. The EPA (1992) recommends assigning a value of one half the detection limit for all results with nondetections as part of the upper confidence limit calculations. This methodology was not used in the BRA because detection limits were not available for all of the sampling analyses. Assigning a zero value to all nondetections allowed the upper confidence limits to be calculated consistently for all of the sampling results.

Data evaluation tasks that were completed as part of the BRA are as follows:

- Identification of sites and co-located facilities that require further evaluation. Co-located facilities are defined as operating or inactive facilities that have the potential for producing future releases of hazardous substances (see Section 3.3).

- Screening of sites to identify the sites with the potential to produce adverse human health (see Section 3.4).

- Review of available sampling data for the retained sites. This review included a "process knowledge" evaluation designed to identify any contaminants that may have been released at a given site but not targeted for sampling and analysis.

- Identification and screening of contaminants detected at each retained site to identify COPCs for quantitative risk assessment (see Section 3.4).

- Identification of potential exposure routes for each COPC.

- Development of each data set for use in the risk assessment.

### 6.1.2 Exposure Assessment

The process of exposure assessment quantifies the receptor intake of COPCs for select pathways. The assessment consists of estimating the magnitude, frequency, duration, and exposure route of chemicals to receptors. The following exposure assessment tasks were performed as part of the BRA:

- Identification and characterization of exposed populations

- Identification of complete exposure pathways

- Estimation of contaminant concentrations at the points of exposure for the following exposure pathways:

- Soil pathway
- Air pathway
- Groundwater pathway

- Estimation of human intake rates
- Calculation of intake factors.

The conceptual site models (CSMs) used to develop the BRA exposure assessment for residential and occupational scenarios are presented in Figures 6-1 and 6-2. The preliminary conceptual site model presented in the WAG 5 Work Plan (DOE-ID 1997) was refined for the comprehensive BRA and the residential and occupational scenarios were separated and illustrated in two figures for clarity. Additional refinements incorporated into the CSMs include the following:

- The contaminant source is given as a soil depth or disposal well rather than the activity that contributed the contamination.
- Soil scenarios for ingestion and dermal contact are grouped together and include the intrusion release mechanism.
- The biotic transport release mechanism was removed from the model because most contaminant exposures calculated in the RI/BRA were based on average soil concentrations that were measured in the depth interval from 0 to 10 ft. In general, plants and animals at the WAG 5 sites would not come into contact with soils that are at depths greater than 3 m (10 ft) below ground surface. Therefore, biotic uptake generally will not affect the average concentrations used to calculate site exposures. A discussion of the qualitative uncertainty associated with eliminating biotic transport appears in Section 6.5.2.

### 6.1.3 Conduct Toxicity Assessment

Toxicity assessment is the process of characterizing the relationship between the dose or intake of a substance and the incidence of an adverse health effect in the exposed population. Toxicity assessments evaluate results from studies with laboratory animals or from human epidemiological studies. These evaluations are used to extrapolate from high levels of exposure, for which adverse effects are known to occur, to low levels of environmental exposures, for which effects can be predicted based on statistical probabilities. The results of these extrapolations are used to establish quantitative indicators of toxicity.

Health risks from all routes of exposure are characterized by combining the chemical intake information with numerical indicators of toxicity. Information used as part of the BRA toxicity assessment is presented in Section 6.3.

### 6.1.4 Risk Characterization

The characterization of risk involves combining the results of the toxicity and exposure assessments to provide a numerical estimate of health risk. This estimate is either a comparison of exposure levels with appropriate toxicity criteria or an estimate of the lifetime cancer risk associated with a particular intake. The nature and weight of evidence supporting the risk estimate, as well as the magnitude of uncertainty surrounding the estimate, also are considered in risk assessment. The results of

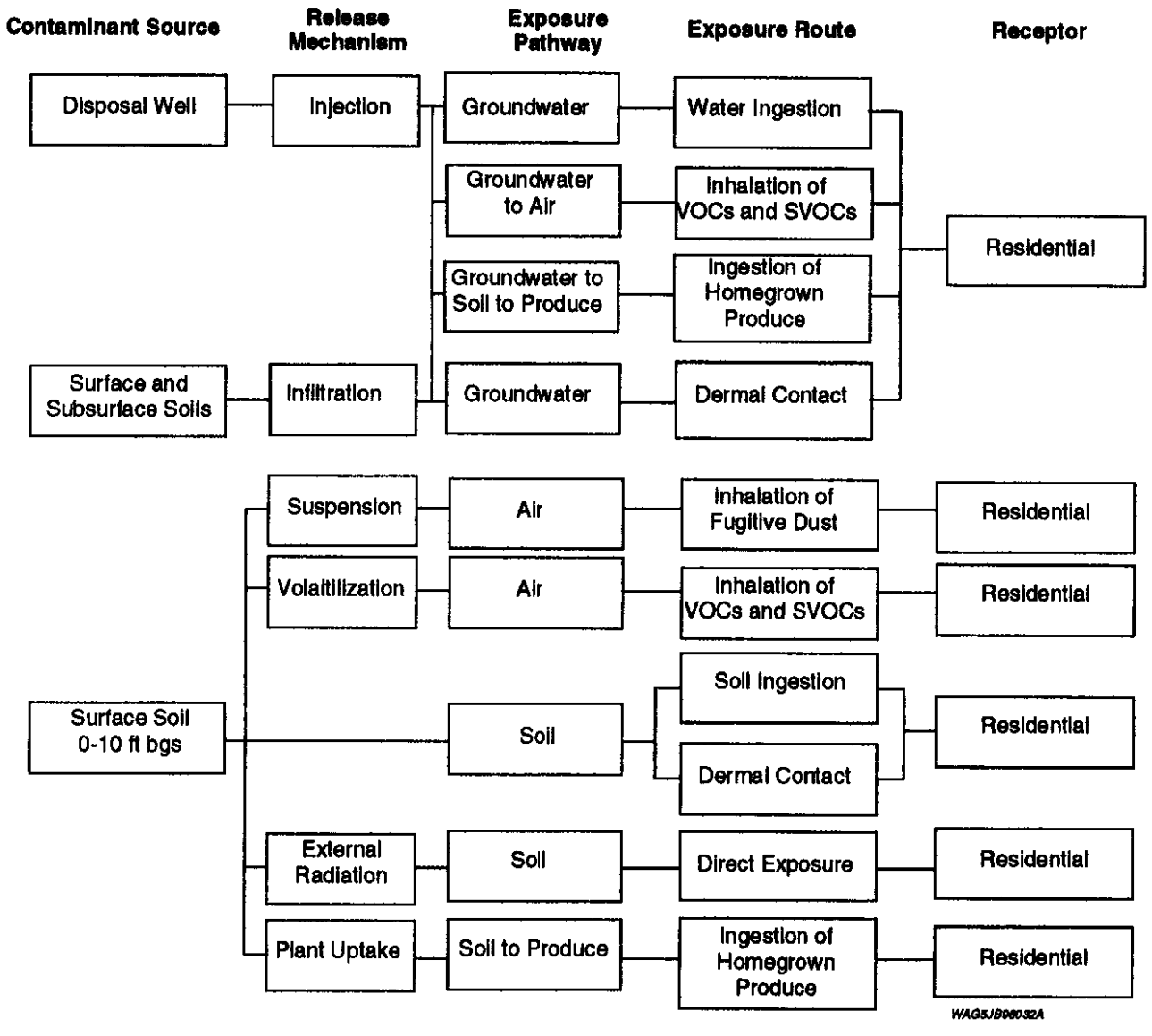


**Figure 6-1.** Human health conceptual site model for the residential scenario.

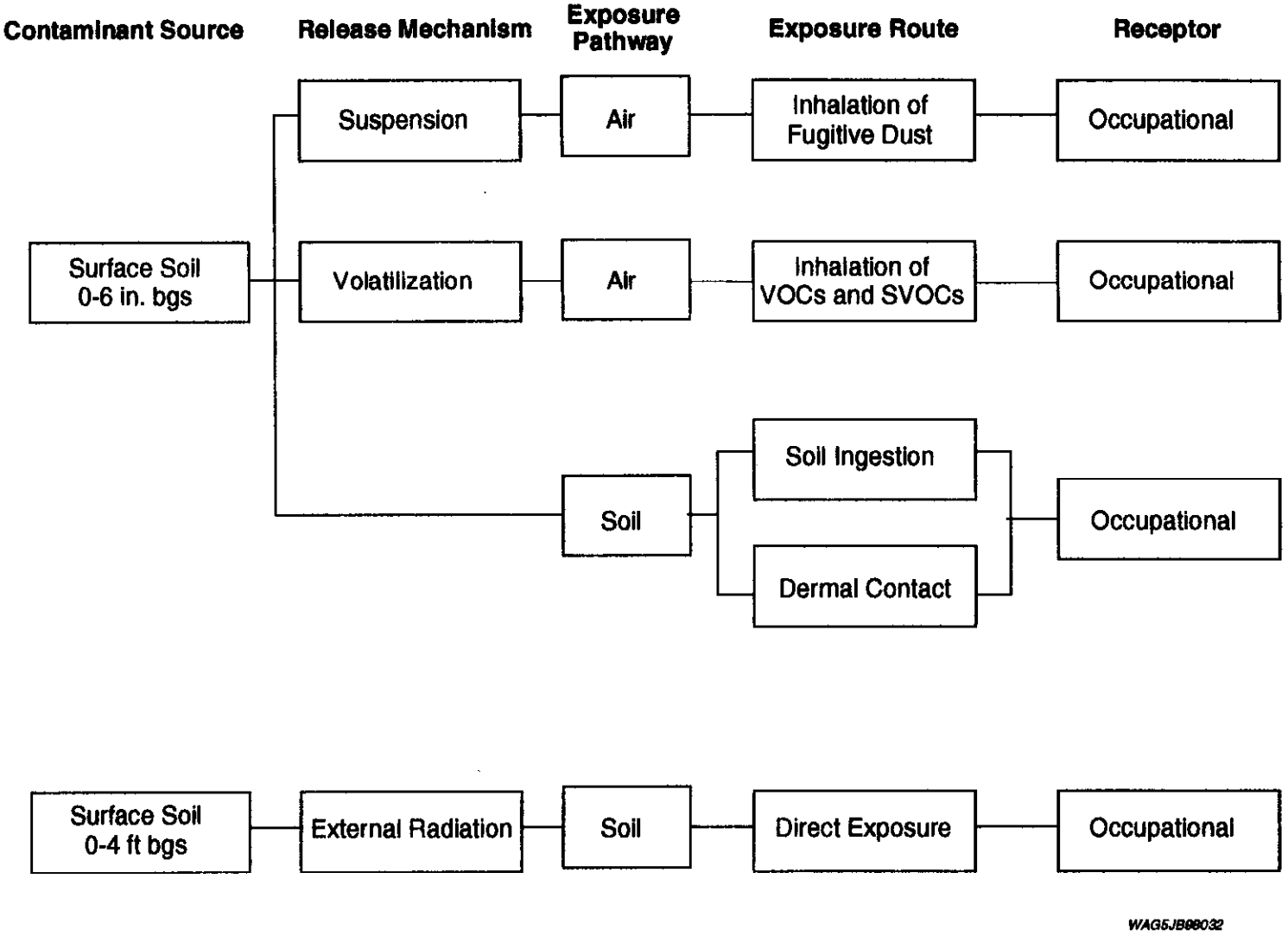


**Figure 6-2.** Human health conceptual site model for the occupational scenario.

the BRA risk characterization process, including risk estimates for each of the retained sites and groups, are presented in Section 6.4 and Appendix B.

## 6.2 Exposure Assessment

In the WAG 5 human health exposure assessment, the receptor intakes of COPCs for select pathways were quantified. The assessment consists of estimating the magnitude, frequency, and duration of COPCs and their exposure route to humans. To quantify the receptor intake, the following activities were performed as part of the BRA:

- Identification and characterization of exposed populations
- Evaluation of exposure pathways
- Estimation of contaminant concentrations at the points of exposure for the following exposure pathways:
  - Soil pathway
  - Air pathway
  - Groundwater pathway
- Estimation of contaminant intakes.

Each of these activities is discussed in the following sections.

### 6.2.1 Identification and Characterization of Exposed Populations

The following human populations potentially could be exposed to contaminants found at, or originating from, WAG 5:

- Workers—Because WAG 5 is currently operational, workers at WAG 5 sites are potential receptors. The following two occupational exposure scenarios are analyzed in the OU 5-12 BRA:
  - A current occupational scenario that lasts for 25 years from the present.
  - A future occupational scenario that starts in 100 years and lasts for 25 years.
- Residents—For the BRA, residential development was considered a potential future use of WAG 5 and a future residential exposure scenario that begins in 100 years was quantitatively evaluated.

Future residents were assumed for the BRA to construct 10-ft basements beneath their homes. Therefore, all contamination detected in the upper 10 ft of each site was evaluated for surface pathway exposures. This analysis method is hereafter referred to as a "residential intrusion scenario," and all residential exposure scenario analyses in the OU 5-12 BRA included the residential intrusion assumption.

However, the residential 10-ft-intrusion scenario was not evaluated for sites that do not have 10 ft of soil (e.g., ARA-01 and ARA-12). Residential risks at these sites were calculated assuming that a future resident would excavate down to basalt and spread the excavated dirt around their home. As a result, the calculated residential risks at the sites are based on average contaminant concentrations down to basalt.

In the residential scenario evaluated in the BRA, a future resident who moves to a retained site in 100 years and lives there for 30 years was evaluated for all exposure pathways except groundwater ingestion, which also was evaluated at the time of peak groundwater concentrations up to 10,000 years in the future. Because the nearest single-family residence is currently located several miles from the boundary of WAG 5, current residents were not simulated in the OU 5-12 BRA.

In general, only adult exposures were evaluated in the residential exposure scenario because of the very conservative assumptions that were incorporated in the BRA calculations. The assumptions most likely caused the calculated risk results to overestimate the actual risks to even sensitive subpopulations, such as children, that would result from exposure to the site contamination.

The exception to evaluating only adult exposures, however, is associated with the soil ingestion exposure route described in Section 6.2.3.1. Under this exposure route, 6 years of childhood soil ingestion and 24 years of adult soil ingestion were included in the contamination intake calculation. Soil ingestion is the most critical exposure route for children who may someday live at WAG 5 because of the relatively large amount of soil that children can ingest.

Groundwater pathway risks were estimated at 100 years in the future for the 100-year residential exposure scenario. Maximum groundwater risks for each COPC within 10,000 years also were estimated. See Section 6.2.3.3 for further discussion of the groundwater pathway analysis.

### 6.2.2 Evaluation of Exposure Pathways

Once potentially exposed populations have been identified and characterized, exposure pathways can be traced from a site to the exposed populations. Each exposure pathway describes a mechanism by which a population or individual could be exposed to contaminants originating from one or more sites within WAG 5. Only exposure pathways deemed to be complete (i.e., those for which a plausible route of exposure can be demonstrated from a site to the receptor) were quantitatively evaluated in the BRA. Based on information presented in the WAG 5 conceptual site model (see Figures 6-1 and 6-2), the following exposure scenarios, exposure pathways, and exposure routes were evaluated in the BRA:

- Exposure scenarios
  - Occupational
  - Residential intrusion
- Exposure pathways
  - Groundwater pathway
  - Air pathway
  - Soil pathway

- Exposure routes
  - Soil ingestion
  - Inhalation of fugitive dust
  - Inhalation of volatiles
  - External radiation exposure
  - Dermal absorption from soil (the exposure route is shown to be screened from further consideration in Section 6.2.3.1)
  - Groundwater ingestion (residential scenario only)
  - Ingestion of homegrown produce (residential scenario only)
  - Dermal absorption of contaminants in groundwater (residential scenario only)
  - Inhalation of volatiles from indoor use of groundwater (residential scenario only).

### 6.2.3 Estimates of Contaminants of Potential Concern Concentrations at Points of Exposure

Exposure concentrations associated with each COPC have been estimated for groundwater, air, and soil. The following sections provide details on how the estimated concentrations for each of these media are calculated.

Two tables that provide general information associated with estimating COPC concentrations at the points of exposure are included in Appendix B. The surface areas and volumes used in the BRA contaminant concentration calculations are shown in Table B-19, and the values assigned to each soil pathway and groundwater pathway parameter (e.g., molecular weight, radionuclide half-life, soil-to-water partition coefficient [$K_d$], solubility, octanol-water partition coefficient [$K_{ow}$], organic carbon partition coefficient [$K_{oc}$], diffusivity, Henry's law constant, and plant uptake factor [PUF]) are shown in Table B-21.

The depths of contamination evaluated for the exposure routes discussed in the following sections are based on guidance given in the INEEL Track 2 manual (DOE-ID 1994). Specifically, contaminant concentrations are based on the 95% upper confidence level (UCL) concentrations of the mean (or the maximum concentration if the maximum is less than the 95% UCL) of samples collected over the following depth ranges indicated in Table 6-1.

The average concentrations for each of the above depth intervals were calculated by averaging detected concentrations in 1-ft intervals. For example, the average concentrations for the depth from 0 to 4 ft were calculated by determining the 95% UCL on the mean or the maximum concentration, whichever was less, for each contaminant in the intervals at 0 to 1 ft, 1 to 2 ft, 2 to 3 ft, and 3 to 4 ft. The four concentrations were then averaged to give the mean concentration for the depth from 0 to 4 ft. For all depth ranges, 95% UCL concentrations of the mean were calculated as described in EPA (1992). Lognormal distributions were assumed for all sampling data sets in these calculations. The soil

**Table 6-1.** Depths of contamination evaluated for the exposure routes.

| Depth | Exposure Routes |
|---|---|
| 0 to 6 in. | Occupational scenario: soil ingestion, inhalation of fugitive dust, inhalation of volatiles |
| 0 to 4 ft | Occupational scenario: external radiation exposure |
| 0 to 10 ft | Residential scenario: all soil pathway and air pathway exposure routes |
| All sample results at all depths | Residential scenario: all groundwater pathway exposure routes |

concentrations used in the BRA calculations, by depth interval, for each COPC are shown in Table B-22 in Appendix B. The concentration values shown indicate that a given COPC was detected in the depth interval shown in the table, not that the COPC contamination extends to the bottom of the interval. For example, chromium could have a calculated concentration for the depth from 0 to 10 ft depth at a given site shown even if the site's chromium contamination only extends from 0 to 5 ft.

In the exposure point concentration calculations, the only form of contaminant degradation considered was radioactive decay (i.e., nonradionuclides are assumed to persist indefinitely in the environment). Radioactive decay is accounted for by estimating radionuclide concentrations at the start of a given exposure scenario, and then calculating the average concentrations that will exist during the length of the scenario. For example, the concentration of a given radionuclide analyzed in the current occupational exposure scenario is the average concentration that would exist between 0 and 25 years in the future and the concentration analyzed in the 100-year future residential scenario is the concentration that would exist from 100 to 130 years. The average radionuclide concentrations over each time period are shown in Tables B-23 through B-27 in Appendix B. The concentrations shown in these tables were used to calculate intakes for radionuclides as discussed in Section 6.2.4.

The effects of radioactive progeny were considered only by using "+D" slope factors (SFs) in the radionuclide risk calculations (see Section 6.4). Decay and ingrowth calculations were not performed for complete radionuclide decay chains. The use of "+D" slope factors accounts for risks produced by daughter products that are in secular equilibrium with their parent radionuclides (EPA 1994).

***6.2.3.1 Soil Pathway Methodology.*** Site-specific soil pathway risks were evaluated for each retained site. The following exposure routes were evaluated in the soil pathway analysis:

- Soil ingestion
- Ingestion of homegrown produce (residential scenario only)
- External radiation exposure
- Dermal absorption from soil (as indicated below, the exposure route is insignificant for all COPCs and is not quantitatively evaluated in the BRA).

***6.2.3.1.1 Soil Ingestion Methodology***—Because exposures through the soil pathway are not likely to occur from more than one site at a time, the soil pathway was evaluated on a site-by-site basis. The possible exception to this rule is associated with the external radiation exposure route.

Retained sites that have radionuclide contamination were evaluated to determine whether radiation produced by one site could affect a receptor located at an adjacent site (see Section 6.5).

As for groundwater and air pathways, soil pathway risks and hazard quotients were calculated at 0 and 100 years in the future for the occupational exposure scenario and at 100 years in the future for the residential scenario.

***6.2.3.1.2 Homegrown Produce Ingestion Methodology***—The ingestion of the homegrown produce exposure route includes an evaluation of COPC concentrations in plants from both root uptake and irrigation with contaminated groundwater. At each retained site, the total source concentration evaluated in the ingestion of homegrown produce exposure route was calculated by summing the 95% UCL concentration of the mean for a given COPC (or the maximum concentration if the maximum is less than the 95% UCL) with the soil concentration that would result from equilibrium partitioning between soil and groundwater contaminated with the COPC. For example, if a COPC has a concentration of 10 mg/kg at a site and the soil concentration of the COPC that would result from equilibrium partitioning with contaminated groundwater is 5 mg/kg, the total contaminant concentration that would be evaluated for the homegrown produce exposure route is 15 mg/kg. The effects of contaminant leaching (i.e., the removal of contamination from the source zone) from groundwater infiltration through the source zone is ignored in this evaluation.

Homegrown produce concentrations assumed for each COPC are presented in Table B-28 in Appendix B. To evaluate the average soil concentration of radioactive COPCs in soil when irrigating with groundwater, the integrated form of Equation 5.39 in Till and Meyers (1993) is used:

$$C_s(t) = \frac{\frac{\dot{I}_v}{L_i+\lambda}\left(t_e + \frac{e^{-(L_i+\lambda)t_e}}{L_i+\lambda}\right) + \frac{C_{so}}{L_i+\lambda}\left(1-e^{-(L_i+\lambda)t_e}\right) - \frac{\dot{I}_v}{L_i+\lambda}}{t_e} \qquad (6\text{-}1)$$

where

$C_s(t)$ = average concentration of a COPC in soil for the exposure period, $t_e$ (pCi/g)

$\dot{I}_v$ = COPC input rate from irrigation (pCi/g-day)

$L_i$ = leach rate constant $(d)^{-1}$

$\lambda$ = radioactive decay rate constant $(d)^{-1}$

$t_e$ = exposure period [10,950 days (30 years × 365 days/year)]

$C_{so}$ = average concentration of COPC in the top 10 ft of soil at the start of the residential exposure period (pCi/g).

For nonradioactive COPCs, this equation reduces to

$$C_s(t) = \frac{\frac{\dot{I}_v}{L_i}\left(t_e + \frac{e^{-(L_i t_e)}}{L_i}\right) + \frac{C_{so}}{L_i}\left(1-e^{-(L_i t_e)}\right) - \frac{\dot{I}_v}{L_i^2}}{t_e} \; . \qquad (6\text{-}2)$$

The COPC input rate from irrigation is given by the following equation:

$$\dot{I}_v = C_w \times \frac{I_R}{\rho \times T} \tag{6-3}$$

where

$\dot{I}_v$ = COPC input rate from irrigation (mg/kg-d or pCi/g-day)

$C_W$ = average concentration of a COPC in groundwater for the exposure period (mg/L or pCi/L)

$I_R$ = irrigation rate (8.47 L/m$^2$ - days × 90 days/365 days) (Maheras et al. 1994)

$\rho$ = soil density (1.5E+06 g/m$^3$)

$T$ = thickness of root zone (0.2 m) (IAEA 1994).

The leach rate constant is given by the following equation (Baes and Sharp 1983):

$$L_i = \frac{P}{\theta_c \times \left(1 + \frac{K_d \times \rho}{\theta_c}\right) \times T} \times CF \tag{6-4}$$

where

$P$ = net water percolation rate (0.86 m/year) [infiltration rate of 0.1 m/year as presented in DOE-ID (1994) plus the contribution from irrigation]

$\theta_c$ = volumetric water content in source volume (0.41 m$^3$/m$^3$) (Rood 1994)

$K_d$ = COPC-specific soil-to-water partition coefficient (cm$^3$/g)

$\rho$ = soil density (1.5 g/cm$^3$)

$T$ = thickness of root zone (0.2 m) (IAEA 1994)

$CF$ = conversion factor (1 year/365 days).

The radioactive decay constant is given by the following equation:

$$\lambda = \frac{ln\,2}{T_{1/2}} \tag{6-5}$$

where

$T_{½}$ = the half-life of a radionuclide (days).

Plant uptake of contaminants through the roots is dependent on the soil concentrations and the plant uptake value ($B_v$) for a given contaminant. Plant uptake of contaminants is calculated using the following equation (EPA October 1995):

$$C_r = C_s(t) \times B_v = C_s(t) \times B_v \tag{6-6}$$

where

$C_r$ = concentration of a COPC in plants (pCi/g or mg/kg)

$C_s(t)$ = average concentration of a COPC in soil over the time interval $t$ (pCi/g)

$B_v$ = contaminant-specific soil-to-plant uptake value (mass of COPC/dry mass of plant material per mass of COPC/dry mass of dry soil)

***6.2.3.1.3 External Radiation Exposure Methodology***—For the external radiation exposure route, standard EPA protocols were used to estimate risks for all retained sites. External radiation exposure risks were calculated by multiplying radiation intakes for specific isotopes by the radionuclide slope factors presented in EPA Health Effects Assessment Summary Tables (HEAST) (EPA 1994). The standard EPA protocols are used because all of the retained sites in the BRA have radionuclide contamination that is at least 6 in. thick over a large area. This thickness is large enough to satisfy the assumption that an increase in source thickness will not cause an increase in surface radiation exposures.

***6.2.3.1.4 Dermal Exposure Methodology***—Risks from dermal absorption from soil are controlled by the potential for a contaminant to be absorbed through skin. The potential is quantified by a contaminant's dermal absorption factor (i.e., the fraction of a given contaminant that can be absorbed through skin) (ABS). The ABS values are not well quantified for many of the contaminants that have been detected at WAG 5; however, EPA Region 3 has issued general guidelines for default ABS values (EPA December 1995).

Organic contaminants have the greatest potential of the WAG 5 contaminants for producing unacceptable dermal absorption from soil exposures. The reason for this distinction is that organic contaminants have relatively high ABS values. For example, EPA (December 1995) recommends assuming an ABS value of 3% for volatile organic contaminants (VOCs) with vapor pressures lower than benzene (i.e., vapor pressure of less than 95.2 mm mercury), and an ABS value of 10% for semivolatile organic contaminants (SVOCs). Because ABS values are poorly defined for most contaminants and organic contaminants have the greatest potential for producing unacceptable dermal absorption risks, organic contaminants were quantitatively evaluated under the dermal absorption from soil exposure route in the BRA.

The exception to the above rule involves the evaluation of arsenic. Use of an arsenic ABS value of 3.2% is recommended by EPA (December 1995). Because the ABS value is relatively high and arsenic was detected significantly above background values at a limited number of sites, arsenic is included in the exposure analysis.

The following equation is recommended by the EPA (December 1995) for calculating absorbed dose from dermal contaminants:

$$AD = \frac{C_{soil} \times SA \times AF \times ABS \times EF \times ED \times CF}{BW \times AT} \quad (6\text{-}7)$$

where

$AD$ = absorbed dose (mg/kg-day)

$C_{soil}$ = contaminant soil concentration (mg/kg)

$SA$ = skin surface area available for contact (3,000 $cm^2$/event)

$AF$ = soil-to-skin adherence factor (0.5 $mg/cm^2$)

$ABS$ = absorption factor (unitless)

$EF$ = exposure frequency (350 events/year for residential exposures)

$ED$ = exposure duration (30 years for residential exposures)

$CF$ = conversion factor (1E-06 kg/mg)

$BW$ = body weight (70 kg)

$AT$ = averaging time [10,950 days for noncarcinogens (30 years)( 365 days/year)], or [25,550 days for carcinogens (70 years)( 365 days/year)].

Absorbed dose for the dermal absorption exposure route is similar to contaminant intakes for other exposure routes (see Section 6.4). Therefore, risks and hazard quotients for dermal absorption exposures can be calculated using the following equations:

$$Risk = \frac{AD \times SF}{GI} \quad (6\text{-}8)$$

where

$Risk$ = contaminant-specific carcinogenic risk (unitless)

$AD$ = absorbed dose (mg/kg-day)

$SF$ = contaminant-specific oral slope factor $[(mg/kg\text{-}d)^{-1}]$

$GI$ = gastrointestinal absorption efficiency factor [=0.05 (unitless)](EPA 1989)

$$HQ = \frac{AD}{RfD \times GI} \quad (6\text{-}9)$$

where

$HQ$ = contaminant-specific noncarcinogenic hazard quotient (unitless)

*AD* = absorbed dose (mg/kg-d)

*RfD* = contaminant-specific oral reference dose (mg/kg-d)

*GI* = gastrointestinal absorption efficiency factor [=0.05 (unitless)] (EPA 1989).

All of the dermal contaminants that have been identified in WAG 5 surface soil sites are listed in Table B-29 in Appendix B. The table shows for each contaminant the sites at which the contaminant has been detected, vapor pressure, whether each contaminant is a VOC or an SVOC, the assumed ABS value, and whether each contaminant has available oral toxicity information that can be used to calculate dermal absorption risks. As shown in the table, Aroclor-1254, bis(2ethylhexyl)phthalate, arsenic, and diethylphthalate are the only contaminants with calculated dermal absorption from soil risks or hazard quotients.

The soil concentrations for these contaminants and the associated slope factors, reference doses, and estimated dermal absorption risks and hazard quotients are shown in Table B-30 in Appendix B. Potential impacts from dermal exposure to contaminants that do not have well defined ABS values and exposures to contaminants that do not have available toxicity information are discussed further Section 6.5.

***6.2.3.1.5 Soil Pathway Assumptions***—The BRA soil pathway analysis incorporated the following assumptions:

- With the exception of the external radiation exposure route, cumulative impacts from soil pathway exposures from multiple sites will be insignificant.
- The likelihood that a future resident will raise meat and dairy products on a residential lot at WAG 5 is negligible based on INEEL guidance for analysis of the food crop ingestion exposure route (LMITCO 1996). Therefore, risks from the ingestion of meat and dairy products were not quantitatively evaluated in the BRA.
- A receptor is present at each retained site for the full exposure duration (30 years for a residential receptor and 25 years for an occupational receptor).

***6.2.3.2 Air Pathway Methodology.*** All retained sites that have contamination in the top 10 ft of soil were assumed for the WAG 5 BRA to have a contaminant source that can be released into the air pathway. The exposure routes that were evaluated as part of the air pathway analysis include the following:

- Inhalation of fugitive dust
- Inhalation of volatiles.

Because contamination from multiple sites may possibly mix together within the air volume above WAG 5, the air pathway was analyzed in a cumulative manner by site groups (see Section 4.1) in the WAG 5 BRA. To perform this cumulative analysis, group-wide average soil concentrations were calculated for each COPC. The concentration of each COPC in the respirable particulate matter above the site group was assumed to equal this average soil concentration. Averaging contaminant concentrations over the site group for the air pathway produces one contaminant-specific risk estimate for each air

pathway exposure route (i.e., for each time period, each air pathway exposure route has the same risk or hazard index at every retained site within the site group [see Section 6.4]).

The following equations are used to estimate airborne contaminant concentrations:

$$C_{air} = CF \times R \times C_{soil} \tag{6-10}$$

where

$C_{air}$ = contaminant concentration in air (mg/m$^3$ or pCi/m$^3$).

$CF$ = conversion from kg to mg for nonradionuclides or g to mg for radionuclides.

$R$ = airborne respirable particulate matter concentration, 0.018 mg/m$^3$. The value is given in Appendix B of the INEEL Site environmental monitoring reports (e.g., Hoff et al. 1993) and represents the arithmetic mean, with 95% confidence interval for the mean, of weekly airborne respirable particulate matter concentrations by the TAN low volume air sampling station.

$C_{soil}$ = site group average contaminant soil concentration (mg/kg or pCi/g) weighted by site area

and

$$C_{soil} = \frac{\sum C_n A_n}{A_T} \tag{6-11}$$

where

$C_n$ = contaminant soil concentration at site $n$ (mg/kg or pCi/g)

$A_n$ = surface area of site $n$ (m$^2$)

$A_T$ = total area of the sites retained in the site group (m$^2$)

$n$ = number of retained sites.

The equation used for estimating concentrations of airborne volatiles is

$$C_{air} = \frac{\sum (C_n / VF_n) A_n}{A_T} \tag{6-12}$$

where

$C_{air}$ = concentration of airborne volatiles in air (mg/m$^3$)

$C_n$ = contaminant soil concentration at site $n$ (mg/kg)

$VF_n$ = volatilization factor [as described in INEEL Track 2 guidance (DOE-ID 1994)] for site $n$ ($m^3/kg$)

$A_n$ = surface area of site $n$ ($m^2$)

$A_T$ = total area of the retained sites in the site group ($m^2$).

The equations produce conservatively high estimates of airborne COPC concentrations because no adjustment is made for dilution of airborne concentrations from dust blown from uncontaminated areas of the WAG.

As with the soil pathway analysis, the air pathway receptor is either a current occupational worker with a 25-year exposure duration or a hypothetical future resident who is exposed for 30 years. Air pathway risks and hazard quotients are calculated at 0 and 30 years in the future for the occupational scenario and at 100 years in the future for the residential scenario. Estimated concentrations of COPCs in fugitive dust and estimated concentrations of volatiles for each time period are presented in Table B-32 in Appendix B.

***6.2.3.2.1 Air Pathway Assumptions***—The BRA air pathway analysis incorporated the following assumptions:

- The concentration of each retained contaminant in the respirable particulate matter above the site group is equal to each contaminant's group-wide average soil concentration.
- The airborne concentration of each retained contaminant is the same at every point inside the group boundaries.
- The air pathway receptor spends the entire exposure duration (25 years for current occupational workers and 30 years for future residents) working or living within the boundaries of the site group.

***6.2.3.3 Groundwater Pathway Methodology.*** To quantify risks for the future residential receptor (no occupational receptor was simulated for this exposure pathway), modeling of contaminant concentrations in groundwater was required. As discussed in Section 5, the GWSCREEN code was applied to evaluate potential contaminant concentrations in groundwater. GWSCREEN also generates risk estimates. For the groundwater pathway analysis, every contaminant that was not eliminated by the contaminant screening process (as described in Section 3.4 and implemented in Tables B-1 through B-18 in Appendix B) was assumed to have the potential for migrating to groundwater, but only anthropogenic sources of contamination were considered in the analysis. The following exposure routes were evaluated as part of the groundwater pathway analysis:

- Ingestion of groundwater
- Dermal absorption of groundwater
- Inhalation of volatiles produced by indoor use of groundwater.

Waste Area Group 5 contains two types of potential sources of groundwater contamination: (1) contamination injected into the vadose zone and (2) contamination that could leach from surface and

near surface soils. These two contaminant sources were evaluated as described in the fate and transport discussion presented in Section 5.

Groundwater concentrations from surface and near-surface sources were estimated using the computer code GWSCREEN (Rood 1994). For each COPC, GWSCREEN produces groundwater concentrations versus time as the code output. From this output, the maximum 30-year average groundwater concentration and the 30-year average concentrations at 100 years in the future are calculated for each COPC. The average concentrations at year 100 are used to calculate groundwater pathway risks for the residential exposure scenario. The maximum average concentrations are used to calculate maximum expected groundwater risks. A period of 10,000 years was considered in the maximum groundwater risk analysis. Therefore, if a COPC is expected to reach a maximum concentration after 10,000 years in the future, maximum groundwater pathway risks for the COPC were not evaluated.

The total mass of each contaminant considered in the GWSCREEN modeling is calculated by summing the contaminant masses from the retained sites. The contaminant mass at each retained site is derived by multiplying the contaminant's 95% UCL concentration of the mean (or maximum concentration if the maximum is less than the 95% UCL) by the mass of contaminated soil at the site. For example, if a contaminant has a 95% UCL concentration of the mean of 5 mg/kg at three sites with dimensions of 10 × 10 × 1 m, the mass of the contaminant that would be used in the GWSCREEN modeling would be

$$2.3E+06\ \text{mg} = (\text{three sites}) \times (5\ \text{mg/kg/site}) \times (10\ \text{m}) \times (10\ \text{m}) \times (1\ \text{m}) \times (1E+06\ \text{cm}^3/\text{m}^3) \times (1.5\ \text{g/cm}^3) \times (1E-03\ \text{kg/g}).$$

Values assigned to various GWSCREEN input parameters are discussed in Section 5. The estimated 100-year groundwater concentrations for each contaminant are given in Table B-33 in Appendix B.

Three input parameters, the length of source parallel to flow, the width of source perpendicular to flow, and the thickness of source, are based on the site dimensions shown in Table B-19 in Appendix B. The length and width values are calculated by taking the square root of the total retained site surface area. The thickness of the source is the average depth of contamination at the retained sites.

Appendix D contains the GWSCREEN output files for each COPC, and the results of the GWSCREEN runs are summarized in Table B-33 in Appendix B. Because the retained site sources are combined for the GWSCREEN modeling, the GWSCREEN output concentrations are not projected to occur at any specific point beneath the WAG. GWSCREEN generates conservative estimates of the maximum groundwater concentrations that might occur at any point beneath the WAG during the residential exposure scenario. Therefore, the contaminant concentrations shown in Table B-33 in Appendix B probably overestimate the true aquifer concentrations that will be produced by infiltration of contaminants at WAG 5.

Because of the great complexity of the subsurface beneath WAG 5 and limited information about factors that influence flow and transport of contaminants in groundwater, the uncertainty about the potential health effects associated with the groundwater pathway exposure routes is greater than the uncertainty associated with any other exposure pathway in the WAG 5 BRA. To compensate for the relatively large uncertainty, conservative assumptions were used throughout the groundwater pathway analysis, as discussed in Section 5. Examples of some of the conservative assumptions that were used in the GWSCREEN analysis are as follows:

- All infiltration into the WAG occurs through contaminated areas of the WAG. No adjustment is made for infiltration into areas between retained sites.

- Contaminant dispersion is negligible. GWSCREEN uses a plug flow model for contaminant transport through the unsaturated zone and does not make adjustments for contaminant dispersion in the unsaturated zone.

- Groundwater flow through fractured basalt in the unsaturated zone occurs very rapidly in comparison to groundwater flow through sedimentary material. This assumption is incorporated into the GWSCREEN modeling by using a depth to the aquifer that is only one-tenth of the total unsaturated zone thickness beneath WAG 5. Using this small depth results in a relatively short unsaturated zone travel time during which radioactive decay can occur. As a result, the GWSCREEN estimates of radionuclide concentrations are expected to be conservatively high.

- All COPC mass contained in surface soils at the WAG contributes to groundwater contamination. For the GWSCREEN modeling, no adjustment is made for loss of COPC mass from mechanisms such as wind erosion, surface water erosion, and contaminant uptake into plants. The only contaminant loss mechanism that is considered in the groundwater pathway evaluation is radioactive decay.

- Estimates of COPC mass that may be transported to groundwater are based on upper-bound estimates of COPC soil concentrations.

- The groundwater receptor takes all drinking water from a well located at the center of the equivalent rectangle's downgradient edge for 30 years.

- All contaminants are uniformly distributed within the groundwater modeling source volume.

***6.2.3.4 Dermal Absorption from Groundwater.*** Exposures to COPCs through dermal absorption of groundwater are controlled by a COPC-specific permeability coefficient of water through skin ($K_p^w$). According to EPA guidance (EPA 1992), if the permeability coefficient for a given COPC is less than 0.1 cm/hour, then the exposure route for dermal absorption from groundwater produces risks that are less than risks produced by the exposure route for the ingestion of the groundwater for that COPC. In the BRA, the default permeability coefficient used for inorganic COPCs was 1E-03 cm/hour. The permeability coefficients for organic COPCs were estimated in the BRA using the following equation (EPA 1992):

$$LogK_p^w = -2.72 + 0.71 LogK_{ow} - 0.0061 MW \qquad (6\text{-}13)$$

where

$K^w_p$ = permeability coefficient of water through skin

$K_{ow}$ = Octanol/water partition coefficient (unitless)

$MW$ = molecular weight (g/mol).

Permeability coefficients for WAG 5 COPCs are shown in Table B-21 in Appendix B. Because many of the organics have permeability coefficients that are greater than the 0.1 cm/hour screening level,

the dermal absorption from groundwater exposure route was quantitatively evaluated in the BRA. Contaminant intakes for this exposure route were calculated using the equations shown in Section 6.2.4.

***6.2.3.5 Inhalation of Water Vapors from Indoor Water Use.*** In the BRA, exposures from inhalation of water vapors from indoor water use were calculated based on experimental data presented in Andelman (1990). A volatilization constant was derived in the study that defines the relationship between the concentration of a contaminant in household water and the average concentration of the volatilized contaminant in air. In the derivation, all uses of household water were considered (e.g., showering, laundering, and dish washing), and certain reasonable assumptions were made in deriving a volatilization fraction. For example, assumptions about water usage for a family of four, the volume of the dwelling, and the air exchange rate were incorporated in the study. Furthermore, the average transfer efficiency weighted by the type of water use was assumed to be 50% (i.e., half of the concentration of each chemical in water will be transferred into air by all types of water uses).

In the BRA analysis of indoor water use, a central tendency value for a COPC's volatilization fraction (6.50E-02 $mg/m^3$ air per mg/L water [Andelman 1990]) was used to develop estimates of COPC airborne concentrations. The airborne concentrations were calculated by multiplying the central tendency value by the COPC groundwater concentrations shown in Table B-33 in Appendix B. The concentrations were then used to develop contaminant intake estimates using the equations shown in Section 6.2.4. The estimates of COPC airborne concentrations from indoor water use calculated for the BRA are shown in Table B-34 in Appendix B.

## 6.2.4 Estimation of Contaminant Intakes

The general equation (EPA 1989) that is used to calculate intakes for most of the WAG 5 BRA exposure routes is:

$$Intake = \frac{C \times IR \times EF \times ED}{BW \times AT} \qquad (6\text{-}14)$$

where

$Intake$ = contaminant intake (mg/kg-day)

$C$ = concentration of a given contaminant in a contaminated media (e.g., soil, air, or water) (e.g., mg/kg, $mg/m^3$, or mg/L)

$IR$ = ingestion rate of the contaminated media (e.g., kg/day, $m^3$/day, or L/day)

$EF$ = exposure frequency (day/year)

$ED$ = exposure duration (year)

$BW$ = body weight (kg)

$AT$ = averaging time (year).

The above equation applies to all exposure routes except exposure to external radiation. For the external radiation exposure route, intakes are calculated using the following general equation:

$$Intake = C \times ET \times EF \times ED \times CF \quad (6\text{-}15)$$

where

*Intake* = radiation intake (pCi-year/g)

*C* = radionuclide concentration in soil (pCi/g)

*ET* = exposure time (hour/day)

*EF* = exposure frequency (day/year)

*ED* = exposure duration (year)

*CF* = conversion factor (1.14E-04 year/hour).

The specific intake factor equations used for the BRA exposure routes are shown in Tables B-35 through B-46 in Appendix B. The results of the BRA intake factor calculations are shown in Tables B-47 through B-67 in Appendix B.

## 6.3 Toxicity Assessment

This section provides the toxicity constants that were used for risk characterization purposes and summarizes toxicological information for the WAG 5 radioactive and nonradioactive COPCs. For this assessment and consistent with EPA guidance (EPA 1989), the toxicity information is summarized for two broad categories of potential effects: noncarcinogenic and carcinogenic. Slightly different methods are applied to estimate potential health risks associated with exposures to carcinogens and noncarcinogens.

The toxicity constants that were used in the BRA were obtained from several sources. The primary source of information is the EPA Integrated Risk Information System (IRIS). The IRIS database contains only those toxicity constants that have been verified by EPA work groups. The IRIS database is updated monthly and supersedes all other sources of toxicity information. If the necessary data are not available in IRIS, EPA Health Effects Assessment Summary Tables (HEAST) (EPA 1994) are used. The toxicity constant tables are published annually and updated approximately twice per year. The EPA HEAST contain a comprehensive listing of provisional risk assessment information that has been reviewed and accepted by individual EPA program offices, but has not had enough review to be recognized as high-quality, Agency-wide information (EPA 1994).

### 6.3.1 Toxicity Assessment for Carcinogenic Effects

The potential for carcinogenic risk is expressed as an estimated probability that an individual might develop cancer from lifetime exposure. This probability is based on projected intakes and chemical-specific dose-response data called cancer slope factors (SFs). Cancer SFs and the estimated daily intake of a compound, averaged over a lifetime of exposure, are used to estimate the incremental risk that an individual exposed to that compound may develop cancer. This estimate is derived using the following equation:

$$Risk = Intake \times SF \quad (6\text{-}16)$$

where

*Risk* = carcinogenic risk (unitless)

*Intake* = contaminant intake (mg/kg-day or pCi)

*SF* = slope factor [$(mg/kg\text{-}day)^{-1}$ or $(pCi)^{-1}$].

Two classes of potential carcinogens are identified at WAG 5 sites: chemical carcinogens and radionuclides, both of which are discussed in the following subsections.

***6.3.1.1 Toxicity Assessment for Chemical Carcinogens.*** Evidence of chemical carcinogenicity originates primarily from two sources: (1) lifetime studies with laboratory animals and (2) human epidemiological studies. For most chemical carcinogens, animal data from laboratory experiments represent the primary basis for the extrapolation. Major assumptions arise from the necessity of extrapolating experimental results across species (i.e., from laboratory animals to humans), from high-dose regions (i.e., levels to which laboratory animals are exposed) to low-dose regions (i.e., levels to which humans are likely to be exposed in the environment), and across routes of administration (i.e., inhalation versus ingestion). Federal regulatory agencies traditionally have estimated human cancer risks associated with exposure to chemical carcinogens on the administered-dose basis according to the following approach:

- The relationship between the administered dose and the incidence of cancer in animals is based on experimental animal bioassay results
- The relationship between the administered dose and the incidence of cancer in the low-dose range is based on mathematical models
- The dose-response relationship is assumed to be the same for both humans and animals if the administered dose is measured in the proper units.

The effects from exposure to high (i.e., administered) doses are based on experimental animal bioassay results while effects associated with exposure to low doses of a chemical are generally estimated from mathematical models.

For chemical carcinogens, the EPA assumes that a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation and tumor induction. This mechanism for carcinogenesis is referred to as stochastic, which means that theoretically any exposure, regardless of how low, to a given chemical poses at least a small, but finite, probability of generating a carcinogenic response.

Because risk at low exposure levels cannot be measured directly either in laboratory animals or human epidemiological studies, various mathematical models have been proposed to extrapolate from high to low doses (i.e., to estimate the dose-response relationship at low doses). The three most frequently used models are (1) the one-hit model, (2) the log-probit model, and (3) the multistage model (Armitage and Doll 1961). The one-hit model is based on the premise that a single molecule of a contaminant can be the single event that precipitates tumor induction (Cornfield 1977)—that is, some finite response is associated with any exposure. The log-probit model incorporates the assumption that a

response is distributed normally with the logarithm of the dose (Mantel et al. 1971). This theory seems to have little scientific basis, though some physiological parameters are lognormally distributed. This model usually yields much lower potency estimates because of the implied threshold at lower doses.

Regulatory decisions are based on the output of the linearized multistage model (EPA 1989). The basis of the linearized multistage model is that multiple events (versus the single-event paradigm of the one-hit model) may be needed to yield tumor induction. The linearized multistage model reflects the biological variability in tumor frequencies observed in animals or human studies (Crump, Guess, and Deal 1977). The dose-response relationship predicted by this model at low doses is essentially linear. Use of this model provides dose-response estimates that are intermediate between the one-hit and the log-probit models. Slope factors calculated for nonradiological carcinogens using the multistage model represent the 95th percentile UCL on the probability of a carcinogenic response. Therefore, risk estimates based on these SFs are conservative and represent upper-bound estimates of risk for which the probability is only 5% that the actual risk is greater than the estimated risk.

Most models produce quantitatively similar results in the range of observable data but yield estimates that can vary by three or four orders of magnitude at lower doses. Animal bioassay data are simply not adequate to determine whether any one of the competing models is better than the others. Moreover, the evidence does not indicate that the precision of low-dose risk estimates increases through the use of more sophisticated models. Thus, if a carcinogenic response occurs at the exposure level studied, it is assumed that a similar response will occur at all lower doses, unless evidence to the contrary exists.

Uncertainties in the toxicity assessment for chemical carcinogens were dealt with by classifying each chemical into one of the following groups according to the weight of evidence from epidemiological studies and animal studies:

Group A—Human carcinogen (sufficient evidence to indicate carcinogenicity in humans)

Group B—Probable human carcinogen (B1–limited evidence of carcinogenicity in humans, and B2–sufficient evidence to indicate carcinogenicity in animals with inadequate or lack of evidence in humans)

Group C—Possible human carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data)

Group D—Not classifiable for human carcinogenicity (inadequate or no evidence)

Group E—Evidence of noncarcinogenicity for humans (no evidence of carcinogenicity in adequate studies).

Table B-21 in Appendix B provides the SFs, in $(mg/kg/day)^{-1}$, and the weight-of-evidence for each WAG 5 COPC.

To obtain an estimate of the total carcinogenic risk from modeled exposures to carcinogens at a site, cancer risks are summed across all exposure routes for all carcinogens. Cancer risks from exposure to multiple carcinogens across multiple pathways are assumed to be additive based on EPA carcinogen risk assessment guidelines (EPA 1986).

***6.3.1.2 Toxicity Assessment for Radionuclides.*** An extensive body of literature exists that describes the health effects of radionuclides on humans and animals. Intensive research by national and international commissions has resulted in the establishment of widely accepted limits to which workers and the public may be exposed without sustaining clinically detectable effects. The EPA has classified all radionuclides as Group A carcinogens because radionuclides emit ionizing radiation, which, at high doses, is associated with increased cancer incidence in humans. Human epidemiological data collected from the survivors of the Hiroshima and Nagasaki bomb attacks form the basis for the most recent extrapolation put forth by the National Academy of Science (BEIR III 1980). Conversely, for most nonradiological carcinogens, animal data from laboratory studies form the primary basis for the extrapolation.

Another fundamental difference between the assessment of potential toxicity associated with exposure to radionuclide and nonradionuclide carcinogens is that SFs for radionuclides are typically best estimates (i.e., mean or median values rather than upper 95th percentile values). Finally, the SFs for radionuclides are expressed in different units—that is, risk per picocurie (pCi) rather than risk per (mg/kg/day) for nonradionuclides.

Table B-21 in Appendix B lists SFs for all radionuclides identified at WAG 5 sites. These nonthreshold SFs account for the following: the amount of radionuclide transported into the bloodstream, the decay of radioactive progeny within the body, the distribution and retention of the radionuclide and its progeny (if any) in the body, the radiation dose delivered to specific organs and tissues, and the age and sex of the exposed individuals (EPA 1994).

### 6.3.2 Toxicity Assessment for Noncarcinogenic Effects

Potential noncarcinogenic effects are evaluated by comparing daily intakes with chronic reference doses (RfDs) developed by the EPA. This section provides a definition of an RfD and discusses how it is applied in the WAG 5 BRA. The RfD values for each of the COPCs identified at WAG 5 sites are provided in Table B-21 in Appendix B.

A chronic RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure that can be incurred during an average lifetime without an appreciable risk of a noncancer effect being incurred in human populations including sensitive subgroups such as pregnant woman and children (EPA 1989). An RfD is based on the assumption that thresholds exist for noncarcinogenic toxic effects (e.g., liver or kidney damage). It is a benchmark dose operationally derived by the application of uncertainty factors of one or more orders of magnitude to doses thought to represent a lowest or no observed adverse effect level in humans. Thus, no adverse effects should be associated with chronic daily intakes below the RfD value. Conversely, if chronic daily intakes exceed the threshold level, some adverse noncarcinogenic health effects potentially could be observed in exposed individuals.

The EPA has not developed RfDs or SFs for the dermal exposure route. In the absence of these factors, the common practice has been to use the available toxicity measures for the oral route of exposure. This approach has been adopted in the WAG 5 BRA.

In evaluating the dermal pathway, the EPA recommends expressing chemical intake as an absorbed dose and adjusting the oral toxicity measures to reflect the absorbed dose (EPA 1989). In deriving such values, consistency is required between the type of dose that forms the basis of the oral toxicity and the type of dose calculated by the dermal exposure models. Specifically, a distinction must be made between an administered dose, or intake (i.e., the amount of chemical taken into the body), and the absorbed dose (i.e., the amount of chemical that crosses the body membranes and enters the bloodstream). Most of the toxicity measures available from the EPA are expressed as administered dose (i.e., intake) rather than

dose at the tissue level (i.e., absorbed dose). The adjustment of the oral toxicity measure can be accomplished only if sufficient data are available in the principal laboratory studies or for the oral absorption efficiency in the species on which the toxicity measures are based. The EPA recommends that exposure estimates for absorption efficiency should not be adjusted if the toxicity values are based on administered doses (EPA 1989).

For risk characterization purposes, potential health effects of chronic exposure to noncarcinogenic compounds are assessed by calculating a hazard quotient (HQ) for each COPC. An HQ is derived by dividing the estimated daily intake by a chemical-specific RfD as shown in the following equation:

$$HQ = RfD / Intake \quad (6\text{-}17)$$

where

*HQ* = hazard quotient (unitless)

*RfD* = reference dose (mg/kg-day)

*Intake* = contaminant intake (mg/kg-day).

An HQ of greater than 1.0 indicates that exposure to a given contaminant (at the concentrations and for the duration and frequencies of exposure estimated in the exposure assessment) may cause adverse health effects in exposed populations. However, the level of concern associated with exposure to noncarcinogenic compounds does not increase linearly as HQ values exceed 1.0—that is, HQ values do not represent a probability or a percentage. For example, an HQ of 10 does not indicate that adverse health effects are 10 times more likely to occur than an HQ value of 1.0. Two conclusions only are appropriate: (1) an HQ value greater than 1.0 indicates that noncarcinogenic health impacts are possible and (2) the higher the HQ value exceeding unity, the greater the concern about potential adverse health effects.

Consistent with Superfund risk assessment guidance (EPA 1989), chemical-specific HQs are summed across exposure routes to calculate a hazard index (HI) for each COPC. Individual pathway HI values are then summed to determine a cumulative HI value for all exposure pathways and COPCs at each site. This approach may result in a situation in which a total HI value for a given site may exceed unity even though none of the chemical-specific HQ values at the site exceeds unity.

### 6.3.3 Toxicity Profiles

The following subsections present general and contaminant-specific information on health effects relating to the COPCs evaluated in the WAG 5 BRA. All information presented in this section is from the EPA IRIS database unless otherwise specified. Chemical-specific toxicity values for each COPC discussed in this section are presented in Table B-21 in Appendix B.

#### *6.3.3.1 Organic Contaminants.*

***6.3.3.1.1 1,4-Dichlorobenzene***—The isomer 1,4-dichlorobenzene is used primarily as an air deodorant and an insecticide, which account for 90% of its total production.

Poison by ingestion and possibly other routes, 1,4-dichlorobenzene is moderately toxic by inhalation. Exposure to 1,4-dichlorobenzene can cause liver injury in humans. It is an experimental carcinogen and mutagenic data exist (Sax and Lewis 1987).

Human exposure to dichlorobenzene is reported to cause hemolytic anemia and liver necrosis, and 1,4-diclorobenzene has been found in human adipose tissue. In addition, the dichlorobenzenes are toxic to nonhuman mammals, birds, and aquatic organisms and impart an offensive taste and odor to water. The dichlorobenzenes are metabolized by mammals, including humans, into various dichlorophenols, some of which are as toxic as the dichlorobenzenes. Points of attack are the liver, respiratory system, eyes, kidneys, and skin. Exposure can result in headaches, eye irritations, periorbital swelling, profuse rhinitis; anorexia, nausea, vomiting, weight loss, jaundice, and cirrhosis (Sittig 1985).

***6.3.3.1.2 Di(2-ethylhexyl)phthalate***—The contaminant di-2-ethylhexyl-phthalate (DEHP) is a synthetic chemical that is commonly added to plastics to make them flexible. It is a colorless liquid with almost no odor. It can move out of plastic materials into the environment over long periods and is widespread in the environment. A large amount of plastic that contains DEHP is buried at landfill sites. It does not evaporate easily, and thus very little will be present in the air. When it is released to water, it dissolves very slowly into underground water or surface waters that contact it. Though DEHP dissolves more easily in materials such as gasoline, paint removers, and oils than it does in water, it has been found in groundwater near waste disposal facilities. When DEHP is released to soil, it usually attaches strongly to the soil and does not move very far away from where it was released. It takes many years before DEHP in buried or discarded materials disappears from the environment (ATSDR 1993a).

Routes of entry into the body are inhalation, ingestion, and skin and eye contact. Irritation of the eyes and mucous membranes, nausea, and diarrhea may result from exposure to DEHP (Sittig 1985).

Short-term exposures to high levels of DEHP interfered with sperm formation in mice and rats. These effects were reversible, but sexual maturity was delayed when the animals were exposed before puberty. After long-term exposure to high doses, fertility of both male and female rats was decreased. Studies of pregnant mice and rats exposed to high levels of DEHP indicated adverse effects on the development of the fetus. In addition, long-term exposure of rats to DEHP resulted in structural and functional changes in the kidney (ATSDR 1993a).

Di(2-ethylhexyl)phthalate has been classified as a potential carcinogen (ATSDR 1993a).

***6.3.3.1.3 Diethylphthalate***—Diethylphthalate (DEP) is used as a solvent for cellulose esters, as a vehicle in pesticide sprays, as a fixative and solvent in perfumery, as an alcohol denaturant and as a plasticizer in solid rocket propellants.

The contaminant DEP has few acute or chronic toxic properties and seems to be devoid of any major irritating or sensitizing effects on the skin. Exposure to heated vapors may produce transient irritation of the nose and throat. Conjunctivitis, corneal necrosis, respiratory tract irritation, dizziness, nausea, and eczema are symptoms of exposure to DEP. Diethylphthalate has been shown to produce mutagenic effects Sittig 1985).

***6.3.3.1.4 Phenanthrene***—The phenanthrene is poisonous by the intravenous route. Mutagenic data exist, and it is in experimental neoplastigen and carcinogen. It is moderately toxic by ingestion. It is also a human skin photosensitizer (Sax and Lewis 1987).

***6.3.3.1.5 Polychlorinated Biphenyls***—Aroclor 1242, 1248, 1254, and 1260 belong to the class of compounds known as PCBs. Mixtures of PCBs are classified as Group B2 carcinogens. Data on carcinogenicity in humans following exposures to PCBs are inadequate because of confounding exposures or lack of exposure quantification (EPA 1993). Exposure to commercial PCB mixtures caused hepatocellular cancer in rats and mice, while most genotoxic and mutagenic bioassays with PCBs have been negative. The oral SF for PCBs (as a mixture) is 7.70E+00 $(mg/kg/day)^{-1}$ (EPA 1994).

Polychlorinated byphenyls comprise a physicochemically and toxicologically diverse group of 209 compounds. Their widespread use chemical stability have made them ubiquitous in the environment. The chemical properties of commercial PCB mixtures depend on their degree of chlorination.

The routes of entry of PCBs into the body are inhalation of fume or vapor and percutaneous absorption of liquid, ingestion, and eye and skin contact.

Prolonged skin contact may cause the formation of comedones, sebacious cysts, and pustules, known as chloracne. Irritation of the eyes, nose and throat also may occur. Acute and chronic exposure can cause liver damage.

***6.3.3.1.6 Tetrachloroethene***—Tetrachloroethene is a widely used solvent with particular use as a dry cleaning agent, a degreaser, a chemical intermediate, a fumigant, and medically as an anthelmintic.

Repeated contact may cause a dry, scaly, and fissured dermatitis. High concentrations may produce eye and nose irritation. Acute exposure may cause central nervous system depression, hepatic injury, and anesthetic death. In animal experiments, exposure to tetrachloroethene has produced cardiac arrhythmia and renal injury. Signs and symptoms of overexposure include malaise, dizziness, headache, increased perspiration, fatigue, staggering gait, and slowing of mental ability (Sittig 1985).

Points of attack are the liver, kidneys, eyes, upper respiratory system, and central nervous system (Sittig 1985).

***6.3.3.1.7 Trichloroethylene***—Trichloroethylene is primarily used as a solvent in vapor degreasing. It is also used for extracting caffeine from coffee, as a dry-cleaning agent, and as a chemical intermediate in the production of pesticides, waxes, gums, resins, tars, paints, varnishes, and specific chemicals such as chloroacetic acid.

Trichloroethylene can enter the body by inhalation, percutaneous absorption, ingestion, skin and eye contact.

Exposure to trichloroethylene vapor may cause irritation of the eyes, nose, and throat. The liquid, if splashed in the eyes, may cause burning irritation and damage. Repeated or prolonged skin contact with the liquid may cause dermatitis.

Acute exposure depresses the central nervous system exhibiting such symptoms as headache, dizziness, vertigo, tremors, nausea and vomiting, irregular heart beat, sleepiness, fatigue, blurred vision, and intoxication similar to that of alcohol. Exposure reportedly has resulted in trichloroethylene addiction, peripheral neuropathy, unconsciousness, and death. Alcohol may make the symptoms worse. If alcohol has been consumed, the overexposed worker may become flushed (Sax and Lewis 1987).

Trichloroethylene administered by gastric intubation to mice induced predominantly hepatocellular carcinomas with some metastases to the lungs.

### 6.3.3.2 Anions

***6.3.3.2.1 Chloride***—Chlorides vary widely. Sodium chloride has very low toxicity, while carbonyl chloride (phosgene) is lethal in small doses. When heated to decomposition or on contact with acids or acid fumes, chlorides evolve highly toxic chloride fumes. Some organic chlorides decompose to yield phosgene.

***6.3.3.2.2 Orthophosphate***—High levels of phosphorus, including orthophosphate, are usually related to fertilizer processing and phosphate mining.

Malaise, muscle weakness, dizziness, and sweating are commonly reported early symptoms of orthophosphate poisoning. Headache, diarrhea, nausea, vomiting, abdominal pain, and salivation are often prominent. Reported adverse health effects include miosis, incoordination, and slurred speech. Dyspnea (difficult or labored breathing), bronchospasm, and chest tightness may eventuate in pulmonary edema. Blurred vision, muscle twitching, and spasms characterize some cases. Severe neurologic manifestations, including convulsions, can occur. Bradycardia occurs infrequently from exposure to orthophosphate.

***6.3.3.2.3 Sulfate***—Sulfur dioxide readily oxidizes to sulfate in the atmosphere when catalyzed by transition metals such as iron, manganese, and vanadium in dispersing smokestack plumes or via photochemical processes. During the smelting of metals or the combustion of fossil fuel, sulfuric acid can sorb on ultrafine metal oxide particles and occur as a primary emission. In some coals, for example, as much as 90% of the resident sulfur may be emitted in this form, sorbed on ultrafine ash. Most of the oxidation of sulfur dioxide, however, occurs in the atmosphere. Sulfuric acid and its neutralization products ammonium bisulfate and ammonium sulfate exist typically as fine particulate matter associated with metals in water droplets or on the surface of ash. As such, they may undergo long-range transport to areas distant from the emission source. Because acid sulfates are stronger irritants than sulfur dioxide is and are likely to be encountered in the atmosphere of industrial societies, considerable attention has been directed toward their impact on the pulmonary airways, in which they are likely to be deposited upon inhalation (Klaassen 1996).

***6.3.3.3 Metals.*** Toxicity information for metal COPCs are discussed in the following subsections.

***6.3.3.3.1 Antimony***—Exposure to antimony may occur during mining, smelting and refining, alloy and abrasive manufacture, and typesetting in printing. Antimony is widely used in the production of alloys, imparting increased hardness, mechanical strength, corrosion resistance, and a low coefficient of friction. Pure antimony compounds are used as abrasives, pigments, flame-proofing compounds, plasticizers, and catalysts in organic synthesis. They also are used in the manufacture of tartar emetic, paints, lacquers, glass, pottery, enamels, glazes, pharmaceuticals, pyrotechnics, matches, and explosives. In addition, they are used in dyeing, for blueing steel, and in coloring aluminum, pewter, and zinc.

Antimony and its compounds are generally regarded as primary skin irritants. Lesions generally appear on exposed moist areas of the body, but rarely on the face. The dust and fumes also are irritants to the eyes, nose, and throat, and may be associated with gingivitis, anemia, and ulceration of the nasal septum and larynx.

Antimony metal dust and fumes are absorbed from the lungs into the blood stream. Principal organs attacked include certain enzyme systems, heart, lungs, and the mucous membrane of the respiratory tract. Symptoms of acute oral poisoning include violent irritation of the nose, mouth, stomach, and intestines, vomiting, bloody stools, slow shallow respiration, pulmonary congestion, coma, and sometime death because of circulatory or respiratory failure. Chronic oral poisoning presents symptoms of dry throat, nausea, headache, sleeplessness, loss of appetite, and dizziness. Liver and kidney degenerative changes are late manifestations (Sittig 1985).

***6.3.3.3.2 Arsenic***—Acute exposure to arsenic causes severe throat irritation, gastrointestinal disturbance, and muscle spasms, which may be followed by vertigo, delirium, and coma follow this. Facial edema also may be evident. Sensory loss and hematopoietic symptoms associated with acute exposure are usually reversible. Malaise and fatigue mark chronic exposure, either by ingestion or inhalation. Changes in the skin include hyperkeratosis. Anemia and neuropathy, liver injury, and "blackfoot disease" also result from chronic exposure.

Arsenic is a known carcinogen in humans. Ingestion is associated with increased incidence of skin cancer; lung cancer results from inhalation. Insufficient data exist to determine carcinogenic effects in animals.

The EPA oral SF for arsenic is 1.8E+00 $(m/kg\text{-}day)^{-1}$, and the inhalation unit risk is 2.4E-03 $(g/m^3)^{-1}$. The confidence in the inhalation unit risk is somewhat uncertain because of the confounding variables in epidemiological studies and only one exposure dose was used in the animal studies. Confidence in the oral SF is relatively high because several studies show significant increases in the carcinogenic response.

***6.3.3.3.3 Barium***—Metallic barium is used to remove residual gas in vacuum tubes and in alloys with nickel, lead, calcium, magnesium, sodium, and lithium. Barium compounds are used in the manufacture of litopone (a white pigment in paints), chlorine, sodium hydroxide, valves and green flares; in synthetic rubber vulcanization, x-ray diagnostics work, glassmaking, papermaking, sugar beet purification, and animal and vegetable oil refining. Barium compounds are used in the laying of brick and tile, pyrotechnics, and electronics industries. They are found in lubricants, pesticides, glazes, textile dyes and finishes, pharmaceuticals, and in cements that will be exposed to saltwater. Barium is used as a rodenticide, a flux for magnesium alloys, a stabilizer and mold lubricant in the rubber and plastics industries, an extender in paints, a loader for paper, soap, rubber, and linoleum, and as a fire extinguisher for uranium or plutonium fires.

Routes of entry include ingestion or inhalation of dust or fumes, and skin and eye contact. The points of attack are the heart, lungs, central nervous system, skin, respiratory system, and eyes (ATSDR 1992a).

Alkaline barium compounds may cause local irritation to the eyes, nose throat, and skin. When barium in ingested or given orally, the soluble, ionized barium compounds exert a profound effect on all muscles and especially smooth muscles, markedly increasing their contractility. The heart rate is slowed and may stop in systole. Other effects are increased intestinal peristalsis, vascular constriction, bladder contraction, and increased voluntary muscle tension. The inhalation of the dust of barium sulfate may lead to deposition in the lungs in sufficient quantities to produce "baritosis"—a benign pneumoconiosis (Sittig 1985).

***6.3.3.3.4 Cadmium***—The critical effect of cadmium is a significant amount of protein in the urine. Ingestion of cadmium can cause adverse effects to the kidney, blood, liver at high concentrations, bone, testes in rats, gastrointestinal (GI) tract, and immune and cardiovascular systems. Symptoms from

ingestion of cadmium include nausea, vomiting, salivation, abdominal pain, cramps, and diarrhea. In addition, cadmium may cause reproductive and developmental effects (ATSDR 1993b). Because there is no evidence of cadmium causing cancer through ingestion, it is not considered a carcinogen by the EPA for this route.

Very little cadmium enters the body through the skin. A larger portion (5 to 8%) is absorbed by the intestinal tract (Amdur, Doull, and Klaassen 1991), and approximately 30 to 60% of inhaled cadmium is absorbed by the lungs (ATSDR 1993b), in which it has a biological half-life as long as 30 years (Amdur, Doull, and Klaassen 1991). Iron deficiencies have been observed to increase cadmium uptake in humans and animals (ATSDR 1993b).

In animals, reproductive and developmental effects have been observed (ATSDR 1993b). Several factors affect the absorption of cadmium including age (ATSDR 1993b). The placenta is a partial barrier. Cadmium is present in human milk. Cadmium exposure may increase the risk of prostate cancer (Sittig 1985).

Symptoms from acute inhalation of cadmium fumes initially include coughing, chest pain, sweating, and chills and progress to labored breathing, coughing, and generalized weakness (Sittig 1985). Kidney damage occurs from both inhalation and ingestion exposures. Lung diseases (e.g., severe pulmonary irritation and bronchitis) are caused by high inhalation exposures (ATSDR 1993b). The liver, testes, immune system, nervous system, and the blood are damaged by cadmium exposure, and inhalation has been shown to cause cancer. Tumors are located in the lungs, trachea, and bronchus.

The EPA has published two RfDs for cadmium: one for food ingestion, 1E-03 mg/kg-day, and the other for water ingestion, 5E-04 mg/kg-day. Both of these values come from IRIS. An uncertainty factor of 10 is used for both oral RfDs to account for sensitive individuals. Confidence in the oral toxicity values is high in that they are derived from a toxicokinetic model that applies data from several studies to calculate the absorption, distribution, metabolism, and elimination of cadmium.

The EPA has classified cadmium as a B1 human carcinogen for inhalation and has assigned an inhalation unit risk for cadmium of 1.8E-03 $(g/m^3)^{-1}$. This value should not be used if the air concentration exceeds 6 $g/mg^3$. The confidence in the inhalation unit risk is fairly high because it is derived from human data, and the types of exposure are more realistic for environmental exposures (i.e., cadmium salts versus cadmium fumes and oxides).

***6.3.3.3.5 Chromium***—The IRIS database contains no exposure information on chromium. Chromium exists as an ore in nature and is most widely used in steel manufacturing, plating, paint and pigment manufacturing, and leather tanning. While chromium exists in several valence states, only the trivalent form is an essential element for humans, and plays an important role in glucose and lipid metabolism. A deficiency is characterized by symptoms similar to diabetes mellitus and produces aortic plaques in rats. A deficiency of trivalent chromium also increases the toxicity of lead.

Intake of chromium in all forms occurs monthly through the water supply. The body stores chromium in skin, lungs, muscle, and fat. Hexavalent chromium is toxic, but is converted to trivalent in the skin when present in small concentrations. Exposure to high concentrations of the hexavalent (industrial) form induces dermatitis, inflammation of the larynx and liver when inhaled, and renal tubular necrosis.

The IRIS has listed the no observed adverse effect level (NOAEL) for chromium(VI) as 25 mg/L. The inhalation unit risk applicable to chromium(VI) is 1.2E-02 $\mu g/m^3$. The oral RfD is

5.00E-03 mg/kg/day for hexavalent chromium and 1.0 mg/kg/day for trivalent chromium with an associated uncertainty factor of 100.

High rates of lung cancer have been associated with chromate-producing industry workers. Chromate salts are carcinogenic in rats exposed by inhalation.

***6.3.3.3.6 Cobalt***—Nickel-aluminum-cobalt alloys are used for permanent magnets. Alloys with nickel, aluminum, copper, beryllium, chromium, and molybdenum are used in the electrical, automobile, and aircraft industries. Cobalt is added to tool steels to improve their cutting qualities and is used as a binder in the manufacture of tungsten carbide tools.

Various cobalt compounds are used as pigments in enamels, glazes, and paints, as catalysts in afterburners, and in the glass, pottery, photographic, and electroplating industries.

Cobalt dust is mildly irritating to the eyes and to a lesser extent to the skin. It is an allergen and has caused allergic sensitivity-type dermatitis in some industries in which only minute quantities of cobalt have been used. Inhalation of cobalt dust may cause an asthma-like disease with cough and dyspnea. This situation may progress to interstitial pneumonia with marked fibrosis.

Ingestion of cobalt or cobalt compounds is rare in industry. Vomiting, diarrhea, and a sensation of hotness may occur after ingestion or after the inhalation of excessive amounts of cobalt dust (Sittig 1985).

***6.3.3.3.7 Copper***—Metallic copper is an excellent conductor of electricity and is widely used in the electrical industry in wire for circuitry, coil, and armature windings, high conductivity tubes, commutator bars. Metallic copper is made into castings, sheets, rods, tubing, and wire, and is used in water and gas piping, roofing materials, cooking utensils, chemical and pharmaceutical equipment, and coinage.

Copper compounds are used as insecticides, algaecides, molluscicides, plant fungicides, mordants, pigments, catalysts, and as a copper supplements for pastures, and in the manufacture of powdered bronze paint and percussion caps. They are also used in analytical reagents, paints for the bottoms of ships, electroplating, and solvent for cellulose in rayon manufacture.

Copper salts act as irritants to the intact skin causing itching, erythema, and dermatitis. In the eyes, copper salts may cause conjunctivitis and even ulceration and turbidity of the cornea. Metallic copper may cause keratinization of the hands and soles of the feet.

Industrial exposure to copper occurs chiefly from fumes generated in welding copper-containing metals. The fumes and dust cause irritation of the upper respiratory tract, a metallic taste in the mouth, nausea, metal fume fever, and in some instances discoloration of the skin and hair. Inhalation of dust and fumes, and mists of copper salts may cause congestion of the nasal mucous membranes, sometimes of the pharynx, and on occasion ulceration with perforation of the nasal septum. If the salts reach the gastrointestinal tract, they act as irritants producing salivation, nausea, vomiting, gastric pain, hemorrhagic gastritis, and diarrhea. Chronic human intoxication occurs rarely (Sittig 1985).

***6.3.3.3.8 Lead***—No critical effects of lead have been reported. However, many organs and systems are adversely affected by lead. The major target organs and systems are the central nervous system, the peripheral nerves, the kidney, the GI system, and the blood system (Sittig 1985). Anemia is one of the early manifestations of lead poisoning. Other early effects of lead poisoning can include decreased physical fitness, fatigue, sleep disturbance, headache, aching bones and muscles, digestive

symptoms, abdominal pains, and decreased appetite. The major central nervous system effects can include dullness, irritability, headaches, muscular tremors, inability to coordinate voluntary muscles, and loss of memory. The most sensitive effect for adults in the general population may be hypertension (Amdur, Doull, and Klaassen 1991).

Ingestion and inhalation of lead have the same effects on the human body. Large amounts of lead can result in severe convulsions, coma, delirium, and possibly death. A high incidence of residual damage, similar to that following infections or traumatic damage or injury, is observed from sustained exposure to lead. Most of the body burden of lead is in the bone (ATSDR 1990a). Lead effects in the peripheral nervous system are primarily manifested by weakness of the exterior muscles and sensory disturbances. Lead also has been shown to adversely affect sperm and damage other parts of the male reproductive system (ATSDR 1990a). Dermal absorption of inorganic lead compounds is reported to be much less significant than absorption by inhalation or oral routes of exposure (ATSDR 1990a).

A major concern relative to lead exposure is behavioral effects, particularly in children. Exposure to lead can cause damage to the central nervous system, mental retardation, and hearing impairment in children. Levels of exposure that may have little or no effect upon adults can produce important biochemical alterations in growing children that may be expressed as altered neuropsychological behavior (Martin 1991).

Though the ability of lead to cause cancer in humans has not been shown, the EPA has classified lead as a B2 probable human carcinogen through both ingestion and inhalation routes of exposure. Lead classification is based on the available evidence of cancer from animal studies. Rats ingesting lead demonstrated statistically increased incidence of kidney tumors (ATSDR 1990a). According to some epidemiological studies, lead workers develop cancer, but the data are considered inadequate to demonstrate or refute the potential carcinogenicity of lead to humans. The EPA has not established toxicity values for lead.

***6.3.3.3.9 Manganese***—The IRIS database contains current risk-based exposure information on manganese. Manganese is a human nutrient, serving as an enzyme cofactor supporting basal metabolism in both plants and animals. It is rated as one of the least toxic of the trace elements and is not a human carcinogen. Manganese is more bioavailable in drinking water than in food. Foods with high levels of manganese are cereals, nuts, and leafy green vegetables.

Manganese poisoning occurs as the result of prolonged inhalation of airborne metal-laden dust. This results in neurological and psychological symptoms including irritability, motor impairment, speech disturbances, and compulsive behavior. Prolonged effects are similar to Parkinson's Disease. Very high doses result in nerve degeneration.

***6.3.3.3.10 Mercury***—Mercury could be present in several forms at WAG 5. Each of the different forms of mercury has different effects on the human body. These effects are summarized below for elemental, inorganic, and organic mercury.

***6.3.3.3.10.1*** **Elemental Mercury**—The critical effects of elemental mercury include hand tremors, increases in memory disturbances, and slight subjective and objective evidence of dysfunction of the autonomic nervous system. Harmful effects include coughing, chest pain, dyspnea, bronchitis, pneumonia, tremors, insomnia, irritability, indecision, headaches, fatigue, weakness, stomatitis (inflammation of soft tissues in the mouth), excess salivation, GI disturbance, anorexia, weight loss, proteinuria, and irritation of the eyes and skin (Sittig 1985).

In several studies, death in humans has been reported following acute exposure to high concentrations of metallic mercury vapor. Death is attributed to a loss of respiratory function from severe pulmonary tissue damage (ATSDR 1992b). In addition, workers chronically exposed to low concentrations of mercury exhibited double vision, and acute exposure has caused red, burning eyes, and conjunctivitis (inflammation of mucous membrane of the eye or eyelid) (ATSDR 1992b). In one case, a 13-year-old boy was exposed to mercury vapors for 2 weeks and developed a thyroid enlargement (ATSDR 1992b), but studies of the effect in an occupational setting have not shown the same relationship.

***6.3.3.3.10.2 Inorganic Mercury***—Inorganic mercury is a primary irritant of skin and mucous membranes and has been shown to cause allergic reactions on the skin (ATSDR 1992b). Acute poisoning from mercury vapors primarily affects the lungs (e.g., acute interstitial pneumonitis, bronchitis, and bronchiolitis) (Sittig 1985). Mercury vapor has an affinity for the kidneys. Effects on the kidneys include proteinuria (elevated serum proteins in the urine) (Amdur, Doull, and Klaassen 1991). The critical effect of inhalation of inorganic mercury is the same as that for elemental mercury.

The effects from ingestion of low levels of mercury vary from weakness, loss of appetite, loss of weight, insomnia, indigestion, diarrhea, a metallic taste in mouth, increased salivation, and soreness of the mouth or throat to extreme irritability, excitability, anxiety, delirium with hallucinations, melancholia, and manic depressive psychosis. Acute exposures have been shown to cause severe abdominal cramps, bloody diarrhea, and suppression of urine. Corrosive ulceration, bleeding, and necrosis of the GI tract are usually accompanied by shock and circulatory collapse. If the patient survives notwithstanding the GI damage, renal failure typically occurs in 24 hours. In general, chronic exposure causes increased excitability, tremors, and gingivitis (Amdur, Doull, and Klaassen 1991).

Spontaneous abortions have occurred in women who were exposed to mercury vapors. A significant increase in the number of spontaneous abortions also occurred in women when the fathers were exposed occupationally to mercury vapors. Metallic mercury vapors have been shown to be absorbed through the skin, but the lungs through inhalation absorb the majority of mercury vapor (ATSDR 1992b).

***6.3.3.3.10.3 Organic Mercury***—The critical effect of organic mercury is developmental neurological abnormalities in human infants. The local effect of dermal contact with organic mercury is dermatitis. Systemic effects are to the central nervous system, primarily the brain. Severe poisoning can be fatal or lead to irreversible brain damage resulting in the loss of higher functions (Sittig 1985). Symptoms include numbness and tingling around the mouth, lips, and extremities; a clumsy stumbling gait; difficulty in swallowing and articulating words; a generalized sensation of weakness and fatigue; inability to concentrate; vision and hearing loss; spasticity and tremor; and coma (Amdur, Doull, and Klaassen 1991). Oral ingestion of organic mercury can cause damage to the central nervous system in both fetuses and children (ATSDR 1992b).

The risk evaluation of mercury is based on toxicity values established by the EPA. The ingestion RfD for mercury is 1.0E-04 $mg/m^3$ and is based on methyl mercury (organic mercury). An uncertainty factor of 3 is applied for variability in the human population, in particular the variation in the biological half-life of methyl mercury and the variation that occurs in the hair-to-blood ratio for mercury. In addition, a factor of 3 is applied because of a lack of a two-generation reproductive study and a lack of data for the effect of exposure duration on complications following developmental neurotoxicity effects. The overall uncertainty factor is 10. Confidence in the RfD is medium based on an evaluation of the database and studies included in the database. The inhalation RfC for mercury is 3.0E-04 $mg/m^3$ and is based on elemental mercury. The uncertainty of 30 is based on a factor of 10 to allow for the protection of sensitive human subpopulations and the use of an LOAEL and on a factor of 3 for the lack of a

database—particularly developmental and reproductive studies. Confidence in the RfC is medium based on an evaluation of the database and studies included in the database. Mercury is not evaluated for carcinogenic effects because it is not classifiable for human carcinogenicity.

***6.3.3.3.11 Nickel***—Nickel forms alloys with elements such as copper, manganese, zinc, chromium, iron, and molybdenum. Stainless steel is the most widely used nickel alloy. Permanent magnets are alloys chiefly of nickel, cobalt, aluminum, and iron. Elemental nickel is used in electroplating, anodizing aluminum, casting operations for machine parts, and in coinage; in the manufacture of acid-resisting and magnetic alloys, magnetic tapes, surgical and dental instruments, nickel-cadmium batteries, nickel soaps in crankcase oils, and ground-coat enamels, colored ceramics, and glass. Nickel is used as a catalyst in the hydrogenation of fats, oils, and other chemicals; in synthetic coal oil production; and as an intermediate in the synthesis of acrylic esters for plastics.

Skin sensitization is the most commonly seen toxic reaction to nickel and nickel compounds and is seen frequently in the general population. This often results in chronic eczema. Nickel and its compounds also are irritants to the conjunctiva of the eye and the mucous membrane of the upper respiratory tract.

Elemental nickel and nickel salts are probably carcinogenic, producing an increased incidence of cancer of the lung and nasal passages. Effects on the heart muscle, brain, liver, and kidney have been seen in animal studies (Sittig 1985).

***6.3.3.3.12 Selenium***—Most of the selenium produced is used in the manufacture of selenium rectifiers. It is used as a pigment for ruby glass, paints, and dyes, as a vulcanizing agent for rubber, a decolorizing agent for green glass, and an insecticide; in the manufacture of electrodes, selenium photocells, selenium cells, and semiconductor fusion mixtures; in photographic toning baths; and for dehydrogenation of organic compounds. It also is used in veterinary medicine and in antidandruff shampoos.

Elemental selenium is considered to be relatively nonirritating and is poorly absorbed. Some selenium compounds are strong vesicants and can cause destruction of the skin. They are strong irritants to the upper respiratory tract and eyes, and may cause irritation of the mucous membrane of the stomach. Selenium compounds also may cause dermatitis of exposed areas. Selenium dioxide inhaled in large quantities may produce pulmonary edema.

The first and most characteristic sign of selenium absorption is a garlic odor of the breath. A more subtle and earlier sign is a metallic taste in the mouth. Other systemic effects are less specific: pallor, lassitude, irritability, vague gastrointestinal symptoms, and giddiness. Vital organs appear to escape harm from selenium absorption but, based on the results of animals experimentation, liver and kidney damage are possible (Sittig 1985).

***6.3.3.3.13 Silver***—Humans are exposed to small amounts of silver from dietary sources. The oral intake of silver from a typical diet has been estimated to range from 27-88 μg/day. Over a lifetime, individuals having no excessive exposure accumulate a small but measurable amount of silver.

The critical effect in humans ingesting silver is argyria, a medically benign but permanent bluish-gray discoloration of the skin. Argyria results from the deposition of silver in the dermis and also from silver-induced production of melanin. Though silver has been shown to be uniformly deposited in exposed and unexposed areas, the increased pigmentation becomes more pronounced in areas exposed to sunlight, resulting from photoactivated reduction of the metal. Though the deposition of silver is

permanent, it is not associated with any adverse health effects. No pathologic changes or inflammatory reactions have been shown to result from silver deposition. Silver compounds have been employed for medical uses for centuries. In the nineteenth and early twentieth centuries, silver arsphenamine was used in the treatment of syphilis. More recently it has been used as an astringent in topical preparations. While argyria occurred more commonly before the development of antibiotics, it is now rare.

Toxic effects of silver have been reported primarily for the cardiovascular and hepatic systems. Hepatic necrosis and ultrastructural changes of the liver have been induced by administering silver to vitamin E deficient and selenium deficient rats. Investigators have hypothesized that this toxicity is related to silver-induced selenium deficiency that inhibits the synthesis of the seleno-enzyme glutathione peroxidase.

No evidence of cancer in humans has been reported despite frequent therapeutic use of the compound over the years. It is not classified as to human carcinogenicity. In animals, local sarcomas have been induced after implantation of foils and discs of silver. However, the interpretation of these findings has been questioned in light of the phenomenon of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas (Sittig 1985).

***6.3.3.3.14 Thallium***—Thallium is used mostly in the manufacture of electronic devices, switches, and closures. It also has limited use in the manufacture of special glasses and for medical procedures that evaluate heart disease. Up until 1972, thallium was used as a rat poison, but was then banned because of its potential harm to humans. Thallium is no longer produced in the United States.

The significant, likely routes of exposure near hazardous waste sites are through swallowing thallium-contaminated soil or dust, drinking contaminated water, and skin contact with contaminated soil. EPA Region 3 recommends an oral slope factor equal to 7E-05 $(mg/kg\text{-}d)^{-1}$ for thallium.

***6.3.3.3.15 Vanadium***—Most of the vanadium produced is used in ferrovanadium and of this the majority is used in high speed and other alloy steels with only small amounts in tool or structural steels. It is usually combined with chromium, nickel, manganese, boron, and tungsten in steel alloys. Vanadium pentoxide is an industrial catalyst in oxidation reactions. It also is used in glass and ceramic glazes, as a steel additive, and in welding electrode coatings. Ammonium metavanadate is used as an industrial catalyst, a chemical reagent, and a photographic developer; and in dyeing and printing. Other vanadium compounds are used as mordants in dyeing, in insecticides, as catalysts, and in metallurgy. Because vanadium is itself considered nontoxic, there is little hazard associated with mining; however, exposure to the more toxic compounds, especially the oxides, can occur during smelting and refining. Exposure also may occur in conjunction with oil-fired furnace flues.

Vanadium compounds, especially vanadium pentoxide, are irritants to the eyes and skin. Vanadium compounds also are irritants to the respiratory tract. Entrance to the body is through inhalation of dusts and fumes. More serious exposure may result in pulmonary edema and pneumonia, which may be fatal (Sittig 1985).

***6.3.3.3.16 Zinc***—Zinc is found naturally in the environment and is present in all foods (ATSDR 1988). It is an essential element and occurs in the environment in the 2+ state. Zinc is likely to be strongly sorbed to soil. Relatively little zinc disposed of in landfills is expected to be in a soluble form. Bioconcentration factors of soil zinc by terrestrial plants, invertebrates, and mammals are 0.4, 8, and 0.5, respectively (ATSDR 1988).

Excessive dietary zinc has been shown to cause copper deficiency and anemia (ATSDR 1988). Cadmium also has resulted in the redistribution of zinc to the liver and kidney. Health effects associated with zinc exposure include anemia, liver necrosis, fetal resorption, and, in extreme cases, cessation of reproduction (ATSDR 1988).

***6.3.3.4 Radionuclides.*** The EPA classifies all radionuclides as Group A carcinogens because they emit ionizing radiation and the extensive weight-of-evidence provided by epidemiological studies of radiation-induced cancers in humans. Ionizing radiation has sufficient energy to interact with matter and produce an ejected electron and a positively charged ion. In addition, ionizing radiation can produce new chemical species, free radicals, from water in the body. Free radicals are highly reactive and may combine with other elements or compounds within a cell to produce toxins or otherwise disrupt cellular chemical balance. Such disruptions may result in mutations or other deleterious effects.

Radionuclides are characterized by the type and energy level of the radiation emitted. Radiation emissions fall into two major categories: (1) particulate (e.g., alpha and beta particles) or (2) electromagnetic (e.g., gamma radiation and x-rays).

The general health effects of radiation can be divided into stochastic and nonstochastic effects (i.e., those health effects not related to threshold dose and those related to threshold dose). Developing cancer from exposure to any amount of radiation is a stochastic effect. Examples of nonstochastic effects include acute radiation syndrome and cataract formation, both of which occur only at high levels of exposures.

Radiation can damage cells in different ways. First, radiation can cause damage to the strands of genetic material, DNA, in a cell. The cell may not be able to recover from this type of damage, or the cell may live on in a functionally abnormal condition. If the abnormally functioning cell divides and reproduces, a tumor or mutation in the tissue may develop. The rapidly dividing cells that line the intestines and the stomach and the cells that make blood in the bone marrow are very sensitive to this kind of damage. With doses of 10 to 500 rem, organ damage resulting from the damage the individual cells is reported.. Acute radiation sickness is seen only after doses greater than 50 rem, which usually is received only by personnel in proximity to serious nuclear accidents. Principal adverse effects associated with exposure to ionizing radiation are carcinogenicity, mutagenicity, and teratogenicity.

When cells damaged by radiation are reproductive, genetic damage can occur in the offspring of the person exposed. The developing fetus is especially sensitive to radiation. The type of malformation that may occur is related to the stage of fetal development and the cells that are differentiating at the time of exposure. Radiation damage to children exposed while in the womb is related to the dose the pregnant mother received. Mental retardation is another possible effect of fetal radiation exposure.

The following subsections provide additional information about the specific radionuclide COPCs at WAG 5.

***6.3.3.4.1 Americium-241***—Americium-241 is produced by the beta decay of Pu-241. This isotope has been distributed widely in the environment as a result of nuclear weapons fallout. Americium-241 decays by alpha emission, which makes it an important isotope for internal exposure, whether it is ingested or inhaled. The alpha decay is accompanied by emission of a 60-kilo-electron-volt (keV) gamma-ray with an abundance of 36%. The gamma emission levels are of concern in locations with concentrated Am-241 but are not important at environmental levels. The International Committee on Radiological Protection (ICRP) has assigned a value of 5.00E-04 to $f_1$ for all compounds of americium. For inhalation exposures, the ICRP recommends assigning all compounds of americium to inhalation Group W. Most (90%) of the americium entering the blood stream is deposited in the liver and

the bone, and only a small amount is deposited in human reproductive organs. The biological half-lives in the liver and the bone are 40 and 100 years, respectively. The amount deposited in reproductive organs is considered to remain permanently.

***6.3.3.4.2 Cesium-137***—Cesium-137 is a fission product produced in nuclear reactors and in nuclear weapons detonations. Cesium-137 is rapidly absorbed into the bloodstream and is distributed throughout the active tissues of the body. Metabolically, Cs-137 behaves as an analog of potassium. Its distribution throughout the body and energetic beta and gamma radiation from its daughter, Ba-137m, result in essentially whole-body irradiation (Amdur, Doull, and Klaassen 1991). The radioactive half-life of Cs-137 is 30 years. Its biological half-life in adults is 50 to 150 days, and in children is 44 days. Cesium-137 exists in secular equilibrium with Ba-137m, which is the major contributor to the dose received from a 0.662-megaelectron volt (MeV) gamma ray. The critical organ for Cs-137 exposure is the whole body.

***6.3.3.4.3 Cobalt-58, -60***—Cobalt-60 is an activation product of the irradiation of stable cobalt or nickel in nuclear reactors or nuclear weapons testing. A major component of normal reactor effluent, Co-60 is intensely radioactive, emitting characteristic gamma rays of 1.2 and 1.3 MeV. The radioactive half-life of C0-60 is 5.27 years, but its resident half-life in the human body is only 9.5 days, which is important in limiting the overall exposure from this radionuclide. Cobalt-60 is used in the treatment of cancer.

The critical organ for Co-60 exposure is the whole body. The slope factor is expressed per unit intake as exposure, and is a function of the route of entry.

***6.3.3.4.4 Europium-154, -155***—The radioactive half-lives of Eu-154 and –155 are 8.59 and 4.71 years, respectively. Both behave similarly to Cs-137.

***6.3.3.4.5 Plutonium-238, -239/240***—After inhalation, plutonium may remain in the lungs but can move to the bones and liver (BEIR V 1990). Plutonium generally stays in the body for a very long time and continues to expose the surrounding tissues to radiation (ATSDR 1990b), increasing the probability of carcinogenesis over time. Approximately 50% of the plutonium that enters the blood is retained in the bone and 30% in the liver with retention times of 20 to 50 years (BEIR IV 1988). Inhalation can cause lung tumors in rats, and dermal absorption is limited (BEIR IV 1988).

Plutonium absorption through the GI tract appears to be limited but is increased with decreased iron and calcium levels (BEIR IV 1988). Data have been reported that indicate a much higher GI absorption for certain compounds of plutonium that are unlikely to be encountered in occupational exposures, (e.g. hexavalent plutonium compounds, citrates, and other organic complexes). Absorption also is increased in the very young (ICRP 1978).

Plutonium, which is absorbed into the blood stream, is deposited mainly in the liver and bones (ATSDR 1990b). For dosimetric purposes, all isotopes of plutonium are assumed to be uniformly distributed over all bone surfaces at all times following deposition.

The main source of plutonium in the environment is from nuclear-weapons testing, with smaller contributions from accidents and space power systems burnup in the atmosphere. An estimated 5E-02 pCi/g of plutonium is contained in the top 5 cm of U.S. soil.

Plutonium-238 and Pu-239, -240 have half-lives of 88 years, 2.41E+04 years, and 6.5 E-03 years, respectively, and decay primarily by alpha emission. The alpha particles are accompanied by various low energy X and gamma rays that do not contribute significantly to radiation dose at environmental levels.

***6.3.3.4.6 Radium-226***—Radium is a naturally occurring silvery white radioactive metal that can exist in several isotopes. It is formed when uranium and thorium decay in the environment. It gives off gamma radiation. The half-life of Ra-226 is about 1,600 years.

Exposure to radium is constant because it is present at very low levels in the surrounding environment. Exposure to higher levels of radium can occur to those who live in an area in which it is released into the air from the burning of coal or other fuels. Exposure also results if drinking water is taken from a source that is high in natural radium, such as a deep well, or from a source near a disposal site.

There is not clear evidence that long-term exposure to radium at the levels that are normally present in the environment is likely to result in harmful health effects. Exposure to higher levels of radium over a long period of time may result in harmful effects including anemia, cataracts, fractured teeth, cancer (especially bone cancer), and death. Some of these effects may take years to develop (ATSDR 1990c).

***6.3.3.4.7 Strontium-90***—Strontium is an alkaline earth element and is, therefore, chemically similar to calcium and barium. Strontium follows calcium through food chains from environment to organism, but some degree of discrimination exists against strontium (Kirchman et al. 1993). Strontium-90 is formed during nuclear fission and decays by beta emission. The daughter radionuclide of Sr-90, Y-90, also is radioactive. The radioactive half-life of Y-90 is 64 hours. Yttrium-90 decays by beta emission to the stable isotope Zr-90. Because Sr-90 is produced in the fuel of nuclear reactors, some Sr-90 in the environment at the INEEL may be the result of reactor operations or from waste transported onto the Site. Small amounts of Sr-90 produced in reactor fuel may reach the coolant through defects in the fuel cladding. In coolant purification or following coolant leakage, Sr-90 may reach the gaseous or liquid effluent streams and, in controlled amounts, be released to the environment.

In terrestrial ecosystems, 60 to 80% of Sr-90 from fallout is retained in the upper 5 cm of undisturbed soil (Horne 1978). The rate of movement of Sr-90 in soil typically is slow and depends on soil type; location exchange capacity, rapid water movement, and high electrolyte concentrations increase migration rates. Plants acquire Sr-90 by root uptake and direct deposition.

Strontium-90 has a radioactive half-life of 29.1 years and a biological half-life of 49 years in bone and 36 years in the whole body. The target organ for Sr-90 exposure is the bone marrow. Biologically, it mimics calcium. It emits high-energy beta particles, and its effective dose is high because it concentrates in a relatively small area of the body.

Strontium-90 exists its secular equilibrium with its daughter, Y-90, which decays and also contributes to the dose received by the target organism.

Chronic ingestion of radio-strontium in laboratory animals produces a high incidence of myeloproliferative disease, including frank leukemia, at the highest levels of intake. Chronic intake of radio-strontium results in only a low incidence of bone tumors in swine; however, a high incidence was observed in dogs. Researchers have found that radio-strontium data do not fit the linear dose relationship over a wide dose range and that at low levels the data were fit well by practical threshold or sigmoid response relationships.

***6.3.3.4.8 Thorium-228, -230, -232***—Natural thorium is a radioactive metal. In addition to the natural thorium isotope, Th-232, which occurs at an abundance near 100%, more than 10 other isotopes of thorium exist (ATSDR 1990d).

Thorium is mainly incorporated into the body via inhalation. It is poorly absorbed through the GI tract and approximately 60% of the thorium body burden is present in the skeleton (BEIR IV 1988). In the body, thorium tends to stay where it is first deposited. When injected into humans as Thorotrast, thorium is deposited in the liver, spleen, bone marrow, and lymph nodes (BEIR IV 1988). Because of its deposition in the bone marrow in which red blood cells are formed, thorium-induced anemia has been observed in conjunction with therapeutically administrated Thorotrast. Liver cancers also have been associated with Thorotrast therapy (BEIR IV 1988).

***6.3.3.4.9 Uranium-234, -235, -238***—Natural uranium contains three isotopes: U-234, U-235, and U-238. The percent abundance of each isotope in natural uranium is, respectively, 0.006%, 0.72%, and 99.27% (ATSDR 1990e). Uranium can be found in the earth's crust at an average concentration of 2 ppm. The ambient air concentration of uranium in the United States ranges from 0.3 to 0.011 $fCi/m^3$ (1 fCi = 1E-03 pCi). The concentration in drinking water ranges from 0.07 to 653 pCi/L with a median value of 0.1 to 0.2 pCi/L.

In natural uranium, the radioactivity from U-238 accounts for about half the total radioactivity, and the radiation from U-234 and U-235 accounts for the other half. Uranium emits primarily alpha radiation that is unable to penetrate skin, but can travel short distances in the body if uranium is inhaled or ingested. Because natural uranium emits very small amounts of gamma radiation that can penetrate the skin, little, if any, danger exists from this type of radiation from uranium (ATSDR 1990e). Moreover, no human or animal studies have definitively linked inhalation or oral exposure to natural uranium to the development of cancer.

For the noncarcinogenic health risks associated with uranium, exposure to natural concentrations of uranium in food, water, air, and soil does not appear to have any toxic effects. Animals that have had oral, inhalation, or dermal exposure to large amounts of uranium have developed damage to the kidney tubules, but other systems were not affected.

The only significant systemic health risk in humans from exposure to nonenriched uranium is potential damage to the kidneys. However, epidemiological studies have not noted an increase in deaths from urogenital or renal diseases, and intravenous studies have failed to identify significant damage to human kidneys following exposure to uranium (ATSDR 1990e). Overall, studies in animals and humans also indicate that exposure to uranium is unlikely to produce immunological or neurological effects. Though the data are conflicting, animal studies indicate that exposure to uranium may affect fetal weight and skeletal development in animals, and may possibly alter the ratio of male to female live births in areas in which people have excessive exposure to the metal (ATSDR 1990e). With the exception of soluble salts, no oral or inhalation RfDs are available for uranium on IRIS or HEAST, nor has ATSDR established minimum risk levels for different environmental media (EPA 1994; ATSDR 1990e).

## 6.4 Human Health Risk Characterization

Risk characterization involves estimating the magnitude of the potential adverse human health effects from released COPCs. Specifically, risk characterization involves combining the results of the exposure and toxicity assessments to provide numerical estimates of health risk. These estimates are comparisons of exposure levels with appropriate RfDs or estimates of the lifetime cancer risk with a given intake.

### 6.4.1 Generalized Approach

To quantify human health risks, contaminant intakes are calculated for each COPC by way of each applicable exposure route (see Section 6.2 and Tables B-35 through B-67 in Appendix B). As discussed in Section 5.3, these contaminant intakes are based on measured concentration estimates at each retained site. To determine human health risks, the contaminant-specific intakes are compared to the applicable chemical-specific toxicity data discussed in Section 5.4. The equations that are used to calculate risks for each retained site are discussed in the following subsections.

**6.4.1.1** ***Carcinogenic Health Effects.*** The following calculations are used to obtain numerical estimates, (i.e., unitless probability) of lifetime cancer risks:

$$Risk = Intake \times SF \tag{6-18}$$

where

*Risk* = potential lifetime cancer risk (unitless)

*Intake* = chemical intake (mg/kg/day), or radionuclide intake (pCi)

*SF* = slope factor, for chemicals $(mg/kg/day)^{-1}$, or radionuclides $(pCi)^{-1}$.

The linear low-dose equation shown above is valid at low risk levels (i.e., below estimated risks of 1E-02). In accordance with EPA guidance (EPA 1989), risks that are greater than 1E-02 are calculated using the following one-hit equation:

$$Risk = 1 - exp(-Intake \times SF) \tag{6-19}$$

where

*Risk* = potential lifetime cancer risk (unitless)

*Intake* = chemical intake (mg/kg/day), or radionuclide intake (pCi)

*SF* = slope factor: for chemicals $(mg/kg/day)^{-1}$ or radionuclides $(pCi)^{-1}$.

To develop a total risk estimate for a given site, cancer risks are summed separately across all potential carcinogens at the site as shown in the following calculation:

$$Risk_T = \sum Risk_i \tag{6-20}$$

where

$Risk_T$ = total cancer risk, expressed as a unitless probability

$Risk_i$ = risk estimate for the $i^{th}$ contaminant.

Similarly, risk values for each exposure route are summed to obtain the total cancer risk for each potential carcinogen.

***6.4.1.2 Noncarcinogenic Effects.*** Health risks associated with exposure to individual noncarcinogenic compounds are evaluated by calculating hazard quotients. The hazard quotient is the ratio of the intake rate to the RfD, as follows:

$$HQ = Intake / RfD \tag{6-21}$$

where

$HQ$ = noncancer hazard quotient (unitless)

$Intake$ = chemical intake (mg/kg/day)

$RfD$ = reference dose (mg/kg/day).

Hazard indices (HIs) are calculated by summing hazard quotients for each chemical across all exposure routes. If the HI for any COPC exceeds unity, potential health effects may be a concern from exposure to the COPC. The HI is calculated using the following equation:

$$HI = \sum \frac{Intake_i}{RfD_i} \tag{6-22}$$

where

$HI$ = hazard index (unitless)

$Intake_i$ = exposure level (intake) for the $i^{th}$ toxicant (mg/kg/day)

$RfD_i$ = reference dose for the $i^{th}$ toxicant (mg/kg/day).

In the foregoing equation, intake and RfD are expressed in the same units and represent the same exposure time period.

### 6.4.2 Estimates of Human Health Risk

Estimates of WAG 5 human health risks during each evaluated time period (see Section 6.2 for a discussion of exposure time periods) are presented in Tables B-68 through B-94 in Appendix B. For each time period, carcinogenic risks and noncarcinogenic HIs are shown in separate tables.

As discussed in Section 6.2, risk and hazard index estimates for the air and groundwater pathway exposure routes (i.e., inhalation of fugitive dust, inhalation of volatiles, ingestion of groundwater, dermal absorption of groundwater, and inhalation of water vapor from indoor water use) are calculated in a cumulative manner. As a result, for a given air or groundwater pathway exposure route within a given time period, the risk estimate for the exposure route is the same at every site in a group. The estimated risks and hazard quotients for the occupational and residential scenarios quantitatively evaluated in the BRA are illustrated in Figures 6-3 through 6-8.